MOTOR, STIMULUS AND DRIVE VARIABLES IN ACQUISITION OF ONE-WAY SHUTTLE UNDER UNILATERAL CORTICAL SPREADING DEPRESSION

A Thesis

Presented to

The Faculty of Graduate Studies and Research

University of Manitoba

In Partial Fulfillment

of the Requirements for the Degree

Master of Arts

by

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May 1969

TO MY MOTHER AND FATHER

FACULTY OF GRADUATE STUDIES AND RESEARCH

REPORT OF THESIS EXAMINERS

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Thesis Title: MOTOR, STIMULUS AND DRIVE VARIABLES IN ACQUISITION OF ONE-WAY SHUTTLE UNDER UNILATERAL CORTICAL SPREADING DEPRESSION.

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ABSTRACT

Sensory impairment, motor clumsiness, and disruption of cortical neural associative processes have been implicated as interrelated components in the behavioral effect of spreading cortical depression.

The present experiment attempted to assess the relative contribution of stimulus and motor factors to chemically-induced behavioral impairment in a one-way shuttle setting.

Rate of acquisition of an avoidance habit in unilaterally depressed rats was compared to saline controls in two 2 x 3 x 2 x 2 factorial designs. Combinations of three barrier openings with width varied and three barrier openings with height varied were employed in an attempt to separate effects of stimulus and motor factors. Two shock intensities (1 ma. or 2 ma.) were used to assess the effects of cortical depression on drive level. Performance changes as a function of hemisphere depressed were statistically analyzed for each barrier combination.

Results indicated that acquisition of an aversively reinforced response in depressed rats was significantly dependent on certain dimensions of barrier openings, on drive level, and on side of cortex depressed for barrier openings with height varied.

ACKNOWLEDGEMENTS

I sincerely thank my Committee Chairman, Dr. M. F. Halasz, for advice, guidance and encouragement in this project. I am grateful to Dr. K. R. Hughes for helpful suggestions and comments. Also, I thank Dr. D. F. Shively for useful criticisms and directions.

I am very appreciative of the encouragement and valuable help of Dr. L. Kunc.

I wish to thank Mrs. B. Lough for ready assistance in typing the manuscript.

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CHAPTER I

THE PROBLEM AND INTRODUCTION

STATEMENT OF THE PROBLEM

Whether learning deficits in the spreading cortical depressed (SCD) rat result primarily from impaired motor function or associative disturbances is a complex and confused issue. There appear to be two opposing views to explain learning decrements under depression. Experimenters attribute deteriorated performance to disturbances in motor performance (Tapp, 1962; Moelis, 1963; Winocur, 1965; Schneider, 1965; Freedman & Lash, 1966) or to disruption of cortical associative processes (Bures, Buresova & Zaharova, 1958; Kunc & Kukleta, 1965; Potts & Black, 1966; Russell, Plotkin, & Kleinman, 1968; Plotkin & Russell, 1969). Empirical attempts to separate effects of SCD on motor debility from cortical integrative processes by means of conditioned responses not involving complex skeletomotor reactions have failed to produce consistent results (Papsdorf, Longman & Gormezano, 1965; Mogenson & Peterson, 1966; Hendrickson & Pinto-Hamuy, 1967).

The present experiment attempted to assess the relative influence of SCD on stimulus and motor factors in an aversive conditioning situation. Horizontal and vertical dimensions of the flight opening in a one-way shuttle were varied systematically. Varying widths of the opening, we assumed, would reveal depression-induced deficits in sensory function - possibly in visual acuity, tactile discrimination, kinesthesis, and vestibular function. In the albino rat, almost all direct visual fibers terminate

in the contralateral cerebral hemisphere (Sheridan, 1966). Whether it is monocular blindness or hemianopia that is induced by unilateral functional decortication, we assumed that an impairment in vision (decreased visual fields in monocular blindness or reduced acuity in hemianopia) would contribute to the sensory deficit under unilateral spreading cortical depression (USCD). Wider openings would probably demand less searching for the escape route, while also allowing more area for paw contact with the opening and less motor coordinated movement in negotiating the opening. Narrower openings might also facilitate acquisition - the sides of the opening serving as tactile guides. Increasing the height of the opening, we supposed, would increase the amount of muscular effort of the task, motor impairment presumably interfering most with performance at the highest opening. A differentiating feature between barrier types employed would be the increased effort against gravity necessary to climb through higher openings.

Previous experiments have indicated that strength of UCS, and therefore drive, may also significantly contribute to results obtained (Delprato & Thompson, 1966; Thompson & Enter, 1967). To ensure adequate stimulation, it has been suggested that intensity of shock in the unilaterally or bilaterally depressed rat should be above the 0.9 ma. level (Thompson & Enter, 1967). The present experiment attempted to further study threshold changes to shock during depression by using two UCS levels within the range suggested by prior reports.

In summary, the present experiment attempted to assess the relative extent to which components of motor and perceptual disturbance, at two

levels of drive, contribute to acquisition decrements under USCD.

In an attempt to achieve these objectives, several modifications of the commonly-employed shuttle procedure were indicated by pilot studies. To reliably facilitate acquisition in a single session, pre-CS intervals of 5 seconds in the shock compartment, CS-US intervals of 10 seconds, post-CS intervals of 55 seconds in the safe compartment, and a one-way shuttle procedure were instituted. The procedural modifications made were based on results of previous studies. Page and Hall (1953) suggested that long intervals in the shock compartment may interrupt acquisition. Thompson (1965) reported that short intertrial intervals resulted in poor learning in bilaterally depressed rats. Theios and Dunaway (1964) and Theios, Lynch and Lowe (1966) found that a one-way shuttle setting was a much simpler task for rats to learn. As a result of the method we employed, the acquisition process may have been one of sensitization. However, we sought to investigate variables which may influence acquisition of performance rather than the associative learning process per se.

INTRODUCTION TO THE PROBLEM

The technique of spreading cortical depression (SCD) of Leao (1944) has been extensively used for more than a decade to assess the relative influence of cortical and subcortical regions in acquisition and overt expression of conditioned responses (CRs).

Spreading EEG depression is a slow moving, propagating depolarization of cortical activity evoked by directly acting stimuli such as potassium chloride (Marshall, 1959). Although the mechanisms underlying SCD are as yet unclarified, several investigators have hypothesized that SCD is initiated by a release of potassium ions into the extraneural space in concentrations which depolarize adjacent neurones (Grafstein, 1956; Brinley et al., 1960; Krivanek & Bures, 1960). A high cell density, functional maturation of neurones, and continuity of grey matter appear to be anatomical pre-requisites for the occurrence of SCD (Bures, 1962; Grossman, 1967).

Electrical (Leao, 1944), mechanical (Zachar & Zacharova, 1961), thermal (Zacharova & Zachar, 1961b) or chemical stimulation (Bures & Buresova, 1956) of the cerebral cortex to threshold level produces an almost immediate local decrease in amplitude of the spontaneous electroencephalogram (EEG). After threshold is reached, the local DC shift spreads concentrically from the point of stimulation over the entire cortex at a rate of three to six mm. per minute according to the "all or nothing" principle (Zachar & Zacharova, 1961; Ray & Emley, 1965). The decrease in spontaneous cortical activity and the slow potential change (SPC), a negative shift of the steady potential up to 15 mv,

occur almost simultaneously forming the basic electrophysical changes in SCD (Bures, 1956; Marshall, 1959). Following a refractory period of four to five minutes, slow potential waves can repeatedly spread from the same cortical region. In the interval between each negative swing there is a continuous depression of electrical activity inhibiting the function of cortical cells. Duration of functional decortication may be regulated by the strength of the developing agent, lasting fifteen to twenty minutes after a single wave of depression to several hours (3 hours with a 25% KCl solution).

Liberson and Cadilhac (1953), Liberson and Akert (1955) and Weiss and Fifkova (1960) found that the only subcortical structure susceptible to SCD is the hippocampus. Bures, Buresova, Fifkova, Olds, Olds, and Travis (1962) reported that unilateral spreading depression (USCD) produced a decrease in unit response rate with electrodes placed in the ipsilateral dorsomedial hypothalmus and an increase in unit response with electrodes in the ipsilateral dorsomedial tegmentum. Ochs (1962) demonstrated that bilateral spreading cortical depression (BSCD) depresses the spontaneous electrical activity in the hypothalmus and thalmus. Although the overall activity level does not appear to be altered, the pattern of reticular unit firing is changed (Hendrickson & Pinto-Hamuy, 1967). Changes in subcortical activity induced by SCD have been reported by several experimenters (Bures, 1959; Bures & Buresova, 1960; Rudiger & Fifkova, 1963). Such subcortical changes may interfere with avoidance acquisition (Hjelle & Thompson, 1965).

Spreading depression has never been observed to invade hippo-

campal tissue; hippocampal disturbances do not appear to influence cortical functions (Grossman, 1967). Specificity of SCD is one of its most useful properties. In a functionally hemidecorticated rat, the depression may be limited to one of the hemispheres, thus confining memory traces without disrupting the commisural pathways. Because intracortical connections are probably severed (Bures, Buresova, & Zaharova, 1958) and subcortical structures are influenced (Bures et al., 1961), the function of the nondepressed hemisphere may be altered. Consequently, assessment of the capacity of that hemisphere in learning may be only partial. Bures, Buresova, and Fifkova (1964) reported partial transfer of a passive avoidance response during training under USCD.

Spreading cortical depression is an electrochemical phenomenon accompanied by a general suppression of integrated behaviour. Performance of responses mediated by more complex mechanisms appears more impaired than overt behaviour mediated by more simple mechanisms (Buresova, 1956; Bures, Buresova & Zaharova, 1958).

Altered cortical activity, a product of functional ablation, is reflected in postural and locomotor changes. Whereas BSCD blocks performance of well coordinated and goal directed movements, USCD impairs motor reflexes to a lesser degree (Rudiger & Fifkova, 1963). An absence of any locomotor deficit following USCD has been reported by several investigators (Bures & Buresova, 1960; Rudiger & Bures, 1962; Rudiger, 1962). Subcortically integrated reflexes are almost entirely unaffected by SCD. Corneal and pupillary reflexes remain unchanged in bilaterally depressed rats; spinal flexory reflexes may be exaggerated (Buresova,

1956). Simple postural reactions are apparently undisturbed during BSCD (Buresova, 1956).

Because complicated postural responses are disturbed, it is possible to detect the presence of SCD without electrophysiological indicators (Kunc, 1965). However, motor impairment need not necessarily interfere with acquisition of conditioned responses. Reflexes such as placing, hopping and balancing on a small stand are severely disturbed during depression (Buresova, 1956; Bures et al., 1958; Tapp, 1962; Bures & Buresova, 1960; Mogenson, 1965; Grossman, 1967). The contralateral forelegs of unilaterally depressed rats have been observed to dangle between the bars of the grid floor of the shuttle box and rotor (Mogenson, 1965). General locomotor activity is decreased by SCD. The functionally ablated rat tends to be inactive, slumping on its abdomen (Mogenson, 1965), or assuming a sitting position apparently asleep (Bures et al., 1958). Exploratory activity significantly decreases under USCD (Kunc & Kukleta, 1965). Such changes in spontaneous activity may be indicative of a loss of posture or tonus. Animals able to escape shock exhibit loss of muscle control and stagger over to the goal box (Tapp, 1962). Similarly, performance of pigeons under striatal spreading depression appears to lack perceptual control (Shima, 1964). In contrast with Tapp's observation, Bures (1959) noted that "the posture of the animal and its ability to move are completely undisturbed". Experimental observations generally agree that gross and subtle motor impairment, induced by dural application of potassium chloride, interferes with the expression of motor behaviour. Consequently, the effect of SCD on cortical associative processes is

confounded (Mogenson & Peterson, 1966). Whether performance decrements in learning experiments result from the direct influence of SCD on neural mechanics of learning or from these motor effects is still an unresolved question.

Avoidance acquisition in bilaterally depressed rats has been demonstrated by Bures (1959), Thompson (1964), and Thompson and Hjelle (1965). Travis and Sparks (1963) reported only escape learning; Bures et al., (1958) and Tapp (1962) failed to obtain retention of either escape or avoidance response in animals pretrained without SCD. Avoidance acquisition in unilaterally depressed rats has been demonstrated by Kunc and Kukleta (1965). Their results indicated that, if a USCD rat is able to learn, it acquires slower, extinguishes faster, but has the same relative savings as a normal rat. Lack of uniformity of findings in aversive conditioning studies may be partially attributed to variations in stimuli relevant to acquisition, such as dimensions of the opening, and to different shock levels employed. These procedural modifications obscure the issue of learning under depression.

There appear to be two conflicting viewpoints concerning the nature of the SCD effect. In general, experimenters attribute acquisition impairments in rats to motor deficits due to depression (Tapp, 1962; Moelis, 1963; Mogenson, 1965; Winocur, 1965; Freedman & Lash, 1966), or to "disturbances of cortical mechanisms involved with acquisition" (Bures et al., 1958; Kunc & Kukleta, 1965; Potts & Black, 1966).

Tapp (1962) ascribed loss of the shuttle box avoidance habit in bilaterally depressed rats to "a general loss in ability to perform tasks

involving integrated motor behaviour" as roughly measured by the "stick test". Failure on this test, which required Ss to maintain their balance on a slowly rotating rod, was found to be significantly related to lack of retention of the shuttle response. But the stick test may provide a measure of the integrity of complex postural reflexes which need not form a crucial link in the locomotor pattern of shuttle box avoidance (Russell, Plotkin & Kleinman, 1968). Moelis (1963) found deteriorated motor performance, as indicated by increased time on lever for any bar press and decrements in stable rates of response, in BSCD rats trained to lever press for water on a FR10 schedule of reinforcement. Mogenson (1965) also obtained decrements in a conditioned avoidance paradigm (CAR) using peripheral and cortical stimulation. To avoid grid shock (1 ma.), rats under BSCD or USCD were required to respond to a buzzer or to electrical stimulation of the cerebral cortex or of the basal forebrain region by crossing the center barrier in a shuttle or by rotating a rotor 30^{o}. Mogenson inferred that SD may alter the function of some subcortical structures. Schneider (1965) reported that licking rate, as measured by total amount of water consumed by water-deprived rats over successive three minute intervals of a 30 minute period, is retarded by USCD and abolished by BSCD. His interpretation tends to agree with Tapp's hypothesis of motor debility. Winocur (1965) used a modified Yerkes-Thompson discrimination box to compare the effect of BSCD on performance of two tasks equated for difficulty but varying in motor complexity. In task A, the safe compartment was entered through one of two open doorways; in task B Ss were required to climb through a small window

centrally located about two and one-half inches from the floor. Results indicated greater impairment in response to the more complex task. These findings were also interpreted in terms of a locomotor response decrement hypothesis (Tapp, 1962). In conflict with findings of Winocur, Koranje, Endroczi, and Lissak (1965) reported that rats required to jump onto a small platform 15 cm above floor level showed conditioned escape responding. Since this height is more than twice that of Winocur's small door, it is unlikely bilateral decrements resulted from motor impairment. Freedman and Lash (1966) employed a one-way shuttle in which a guillotine door separated the shock and goal compartments. The authors contended that statistically significant latency increments and rising variances over trials under KCl training are due to "some unspecified motor decrement". However, these results might reflect a learning impairment since latencies increased with practice (Russell, Plotkin, & Kleinman, 1968). The nature of the motor impairment, which has been suggested as the source of learning decrement in depressed animals, has yet to be clearly stated. Culler and Mettler(1934), Girden, Mettler, Finch, and Culler (1936) and Bromiley (1941) showed that surgically decorticated animals perform CRs which involve only gross movements.

Although he argues that SCD has a disorganizing effect on associative processes, Bures (1959, 1960a) obtained results suggesting that depression may profoundly disrupt motor behaviour. When the sensorimotor area of the dominant hemisphere was protected against effects of SCD by $MgCl_2$ while the rest of the cortex was depressed, the rat was able to perform a conditioned motor response. With more diffi-

cult tasks, probably involving greater cortical participation, application of $MgCl_2$ over the motor region afforded less protection. A CR deficit persisted with $MgCl_2$ over cortical areas other than the motor region. These results argue that the motor cortex is important in acquisition of a motor response. Whether it is directly involved in neural associative processes or simply in motor control is as yet undetermined (Winocur, 1965).

On the basis of the experiments cited, it seems evident that impairment of motor function contributes to performance decrements under SCD. To demonstrate that motor debility disturbs learning a causal relationship between them must be shown (Russell, Plotkin & Kleinman, 1968). But neither subjective observation nor the CAR paradigm offers a sufficiently sensitive index of motor decrement (Moelis, 1963); Tapp (1962) showed that S's behavior appeared normal in its emotional reaction to strong shock when a depression effect was observed.

Performance decrements under SCD primarily result from the disruption of cortical associative processes, according to the interpretation of Bures and Buresova (1960). Reduction of the cortical efflux to the reticular system may lower the latter's excitability, disrupting the close interaction between cortical and subcortical neurones apparently necessary for establishing conditioned responses. Because part of the memory forming mechanism is extracortical, SCD may not completely disrupt acquisition.

Results of experiments by Bures (1959), Tapp and Moelis (1961), Kunc and Kukleta (1965), Potts and Black (1966), Russell, Plotkin and

Kleinman (1968) and Plotkin and Russell (1969) suggest that SCD interferes with cortical associative processes rather than motor performance. Bures (1959) demonstrated that primitive conditioned reflexes can be elaborated during SCD. Only 20 to 30 per cent of depressed Ss showed avoidance responding. When a barrier was placed in the box, avoidance acquisition was facilitated in control animals and inhibited in experimental animals. Control Ss showed avoidance behavior and a rapid decrease in escape latencies; depressed Ss showed practically no avoidance behavior and maintained long escape latencies. These results could be accounted for by a gradual recovery from the effects of SCD with time (Tapp, 1962) or by the issue of general motor decrement precluding efficient integration of responses under depression (Moelis, 1963). Brown and Jacobs (1949) and Tapp and Moelis (1961) controlled for possible motor deficits by pairing unavoidable shock with a buzzer. For nondepressed animals who had previously received pairing of a CS with shock, Brown and Jacobs (1949) reported speed of shuttling over a small hurdle increased in learning curve manner. For both depressed and control animals who had experienced pairings of a buzzer with unavoidable shock during acquisition trials, Tapp and Moelis found no evidence of retention and no significant differences between pilot groups. Differences in results might have been due to different shock parameters or to different stimuli used in each study. Brown and Jacobs employed a compound stimulus (light and tone) as CS and a small hurdle between compartments, whereas Tapp and Moelis employed only a buzzer as CS and no barrier between compartments on test trials (Moelis, 1963). Kunc and Kukleta (1965) varied the height of the escape opening

(0 cm or 10 cm) and found acquisition in USCD rats more rapid with the simpler flight route (0 cm opening). According to the view held by Kunc and Kukleta, orientation to the experimental environment, probably a prerequisite for learning, is disrupted by blockade of one hemisphere. As a result of disorientation, animals perform more poorly in the relatively complex environment. Since deterioration in performance was more marked under right hemispheric SCD, Kunc and Kukleta concluded that the right hemisphere is dominant in orienting ability. Winocur (1965) obtained similar results in favor of a motor debility hypothesis. Potts and Black (1966) concluded BSCD blocked the ability of animals to acquire or possibly retain a simple discrimination in which behavior was maintained by secondary stimuli previously paired with intracranial shock. Russell, Plotkin, and Kleinman (1968) found that BSCD selectively blocked start latencies, having no significant effect on running times in acquisition of an avoidance response in the runway. Since motor behavior of BSCD animals was as rapid and as coordinated as of controls, motor deficit did not appear to account for performance in their experiment. Plotkin and Russell (1969) concluded that the effect of intertrial interval is the same for normal and USCD rats in avoidance acquisition in a runway. Quantitative not qualitative deviation from normal during USCD was considered indicative of defective stimulus sampling and encoding or acquisition. Form the above studies it may be inferred that stimulus factors relating to the CS may partly determine whether data seem to support a motor debility or a learning disorganization hypothesis. The complexity of a task may be detected by performance decrements as a

function of hemisphere depressed. In surgically decorticated animals, only the simplest type of conditioned reflexes, such as the general motor escape response, may be elaborated (Pinto-Hamuy, Santibanez & Rojas, 1963). Nonspecific deficits in instrumental learning have been reported in surgical hemidecorticated (Bromiley, 1948) and surgical split-brain preparations (Meikle, Sechzer, & Stellar, 1962; Sechzer, 1964).

Theoretical positions held by Estes (1950), Restle (1955), and Bindra (1961) seem to be in line with a motor impairment explanation of behavior deficit first postulated by Tapp (1962). Estes and Restle stress the importance of sensory events as determiners of a response, the probability of occurrence of a specific response being related to the stimuli perceived by the animal. Pickett (1952) trained rats on elevated and alley-mazes limiting the stimuli to kinesthetic and tactile cues. Results indicated that posterior lesions had no effect on retention while anterior lesions (somatic and sensory areas) disrupted the habit. The authors concluded that removal of stimuli controlling a habit may interfere with learning. Findings of Pickett, therefore, seem to be consistent with arguments of Estes and Restle. Bindra (1961) presents a complementary approach-motor events are of greater importance in a shuttle box situation. Factors which decrease movement delay or preclude elaboration of an avoidance response; Kriekhaus (1965) and Weiss, Krieckhaus, and Conte (1968) found that increased movement preceded improved avoidance performance. Their results support Bindra's formulation.

Theoretical views advanced by Lashley (1929), tend to conform with a learning impairment hypothesis as initially set forth by Bures (1958). Lashley (1929) postulated that learning is controlled by a central autonomous mechanism and that cortical areas are equipotential in their contribution to the association processes. In agreement with this argument, Lashley and Ball (1929) showed that sensory and motor spinal lesions did not impair retention of a maze habit. Since the impairment appears to result from a severe reduction in the mass of functional cortex, Plotkin and Russell (1969) consider the learning deficit under SCD as descriptive evidence for cortical mass action (Lashley, 1929).

Since rats reactivity to sensory stimuli may be decreased by SCD, tasks requiring difficult discriminations and motor responses are probabily more impaired under depression (Thompson, 1964; Winocur, 1965; Thompson & Hjelle, 1965; Mogenson, 1965; Kunc & Kukleta, 1965). Impairment in the ability to make visual discriminations (Bures, 1959; Bures & Buresova, 1960) and motor responses (Tapp, 1962 etc.) as well as freezing (Kunc & Kukleta, 1965) may contribute to longer escape latencies in depressed rats.

During SCD sensitivity to electrical stimulation may be reduced (Marshall, 1959; Delprato & Thompson, 1966; Thompson & Enter, 1967). Results obtained by Delprato and Thompson (1966) suggest that the threshold for shock stimulation under BSCD is raised; 2 ma.-BSCD were inferior in latencies to 0.4 ma. operated controls in a one-way shuttle setting. Thompson and Enter (1967) obtained similar findings using shock intensities of 0.0 to 0.9 ma. in an unconditioned responding situation. The

simplicity of the locomotor response observed during USCD and BSCD in their experiment tends to disagree with a response interference hypothesis (Tapp, 1962).

In a shuttle box setting with nondepressed rats as Ss, Broadhurst and Levine (1963), Moyer and Korn (1964), Levine (1966), and Moyer and Korn (1966) obtained an inverted U-shaped relationship between shock intensity and avoidance acquisition. Broadhurst and Levine (1963) and Levine (1966) found that as shock intensity increased acquisition tended to be disrupted. These authors used shock levels ranging from 0.20 ma. to 0.80 ma. in a modified shuttle box procedure in which interstimulus responses were punished. Moyer and Korn (1964) reported that learning declined after 1 ma. and was maximally disrupted at 2.50 ma. Using a one-way shuttle and UCS intensities ranging from 0.50 ma. to 3.50 ma., Moyer and Korn (1966) reported that high shock levels retarded escape but not acquisition of the avoidance response. Results obtained by Theios, Lynch and Lowe (1966) indicated that rate of conditioning decreased with high-intensity shock in the shuttle procedure but not in the one-way method. "Whether shock intensity will lead to an increment or a decrement in performance is dependent on many factors, among them being the nature of training, the nature of the response, etc." (Levine, 1966).

Performance deficits during SCD may not be entirely accounted for by a general drop in motivation or by motor debility (Mogenson, 1965; Koppman & O'Kelly, 1966). Mogenson (1965) noted that SCD of the ipsilateral hemisphere produced a greater depression of brain self-stimulation

than contralateral hemispheric SCD. Neither a sensory deficit nor a motor disturbance hypothesis is confirmed by Koppman and O'Kelley's (1966) observations of eating behavior in unilaterally depressed rats in a simple T-maze. The authors argued that if USCD resulted primarily in motor impairment, the contralateral side of the mouth would be more depressed than the side ipsilateral to the depression. Also, if a cortical hemianopia were present, limiting its field of vision to the side contralateral to the depression, the rat would probably eat with the side of its mouth ipsilateral to the depressed hemisphere. Koppman and O'Kelly found that USCD rats reliably ate with the side of the mouth contralateral to the depressed hemisphere.

Experiments by Papsdorf, Longman, and Gormazano (1965), Mogenson and Peterson (1966) and Hendrickson and Pinto-Hamuy (1967) attempted to separate the effects of SCD on motor and associative processes by conditioning responses not involving complex skeletomotor reactions. Papsdorf, Longman and Gormezano (1965) assessed the effects of SCD on the classically conditioned nictitating membrane response of the rabbit. KCl blocked the occurrence of CRs, which were spontaneous membrane responses, for about 100 minutes. But the regular occurrence of UCRs, spontaneous membrane responses, in the intertrial interval during SCD suggested that the response was intact. Therefore, the hypothesis of impairment of motor coordination was not confirmed (Tapp, 1962). Mogenson and Peterson (1966) found that depressed animals only exhibited the heart-rate response during the stimulus and not afterward, while the controls exhibited the response both during and after. These results

suggested that the cerebral cortex is not essential for acquisition of the cardiac CR. But sensitization to the vibratory buzzer CS might have influenced their findings. In contrast with results obtained by Mogenson and Peterson (1966), Hendrickson and Pinto-Hamuy (1967) found no retention of a decelerative cardiac CR under neocortical spreading depression.

In summary, the issue of general motor deficit reduces the value of SCD of Leao (1944) as a reversible decortication technique to investigate neural associative processes in un anesthetized animals. Experimental data suggest that the afferent and efferent links of behavior are impaired by SCD-induced disruption of cortical neural activity. Behavior deficit in an aversive conditioning situation may result from disturbance in performance or association; drive level and laterality may significantly contribute to results obtained in USCD studies. The controversy concerning the relative roles of sensory, associative and motor deficits in producing performance decrements might be partially resolved by an attempt to systematically study relevant stimulus and motor factors.

CHAPTER II

THE INVESTIGATION

Subjects

Two hundred naive male albino rats of the Holtzman strain, 60 to 80 days of age, were used in the experiment. Throughout the study Ss were maintained on food and water ad lib and were caged in pairs. One hundred physiological saline controls and 100 USCD animals were randomly assigned to 20 groups with 10 Ss per group according to two 2 x 3 x 2 x 2 factorial designs.

Spreading Depression

Surgery was performed one day prior to testing according to the acute method of Bures (1959). Under light ether anaesthesia, a small midline incision was made, exposing S's skull. A 7 mm opening in each parieto-occipital cortex was then trephined and the skin wound clipped. After a recovery period of approximately 24 hours, the clip was removed and the incision of the unanesthetized rat reopened. In experimental rats unilateral spreading depression was induced by direct dural application of 8 mm squares of filter paper soaked in a 25 per cent KCl solution. (Leao & Morrison, 1945; Bures, 1959). In control animals NaCl (.9 per cent) was applied to the exposed dura in the same fashion.

Apparatus

A stainless steel rod, 3/4 in. in diameter and 17 1/2 in. in length was used to test motor behavior. A modified Lehigh Valley Electronics (LVE) Miller-Mowrer shuttle box of clear Plexiglas was divided into two equal 8 x 8 3/4 in. compartments. The floor of the shock compartment was of

stainless steel grids spaced 2 1/2 cm. apart. The grids could be electrified in a positive, negative, neutral, order by a 1 ma or 2 ma current from a 24 volt LVE constant current shock supply. A removable wooden partition 9 1/2 x 8 in. located behind the barrier separated the shock compartment (right) from the safe compartment (left). Three centrally-located barrier openings labelled 2, 4, 5, with width varied at a constant height of 2 in. (2, 4, or 8 in.) constituted the width parameters. Three barrier openings, designated 1, 2, and 3, with height varied at a constant diameter of 1 in. (0, 2, or 4 in.) were the height parameters employed. The floor of the safe chamber was of cardboard. A Mallory Sonalert Model Sc 628,268-85c tone generator mounted in the center of the safe compartment ceiling delivered a 2.8 kilohertz tone of approximately 82 db at rat level. Each compartment was illuminated by a CGE 509 two volt bulb located in the middle of the rear wall. Shock and tone onset and duration were controlled by a Hunter electronic timer. Intertrial intervals were timed by a stopwatch; latencies were recorded to the nearest .01 second by a Standard timer. Hunter timer and partition were hand operated.

Procedure

All Ss received 3 days adaptation to laboratory conditions. The experimental procedure was identical for all animals, differing only in shock intensity for each barrier. Prior to acquisition, S's motor co-ordination was observed on the rod. Animals capable of grasping and balancing on the rod with forepaws and hindpaws were included in the experiment. Immediately before and immediately after the experimental

session presence of depression was tested by observation of flaccidity of the limbs contralateral to the depressed hemisphere. Five minutes after initial detection of muscular paresis motor coordination was re-tested on the rod to guard against a short-lasting mechanically produced depression. Rats failing to exhibit motor disturbance and Ss with dura damaged surgically were replaced. In control rats (sham operated with NaCl), normal motor coordination was determined by the same procedure as outlined for experimental animals.

Each S was then placed in the right compartment of the shuttle box for an exploration period of 3 minutes. At the end of this period the partition was lowered and S was placed in the safe compartment for a 55 second interval. S was then moved into the right corner of the shock compartment facing the wall opposite the barrier for 5 seconds. The trial began with the onset of the auditory-visual compound CS - simultaneous presentation of the tone and removal of the partition. A CS-US interval of 10 seconds preceded grid shock (1 ma or 2 ma). When S's forepaws touched the floor of the safe compartment or when 60 seconds had elapsed CS and US terminated together. An escape response by S terminated both shock and tone. Crossing the barrier with at least the forepaws in the safe compartment before onset of shock terminated the tone and defined an avoidance response. Latency to avoid or escape, number of reinforcements, and vocalizations were recorded. The experimental session ended upon criterion of 75 trials or 5 consecutive avoidance responses. Control rats were tested for one session. Experimental animals were examined for 2 successive days with each hemisphere depressed followed by a control day (NaCl).

CHAPTER III

RESULTS AND DISCUSSION OF RESULTS

RESULTS

Rate of acquisition of an avoidance response in unilaterally depressed rats was compared with saline controls in two 2 x 3 x 2 x 2 factorial designs. For statistical purposes, combined barriers with height varied (1,2,3) and combined barriers with width varied (4,2,5) were analyzed separately. Barrier 2 was included in both analyses. Analyses of variance were computed according to the Chebib and Becker programme. Mean escape latencies to criterion and mean number of reinforcements to criterion for each S were subjected to statistical analysis. Also, mean escape latencies of trials one to five and trials six to criterion were analyzed to assess the influence of stimuli parameters - depression, barrier dimensions, drive level, and laterality - during early and later stages of acquisition. To meet the criterion of homogeneity of variance, a $\log_{10}$ (x+1) transformation of the data was employed. Results from the first day depression were used in all computations since laterality effects of Day 1 and Day 2 depression did not appear to differ.

A significant depression main effect was consistently obtained for both barrier types (Tables 1-8). Mean escape latencies were significantly longer (Fig. 1 and 2) and more reinforcements were required by depressed rats than saline controls. These results argue that rate of acquisition was significantly retarded in depressed rats in comparison

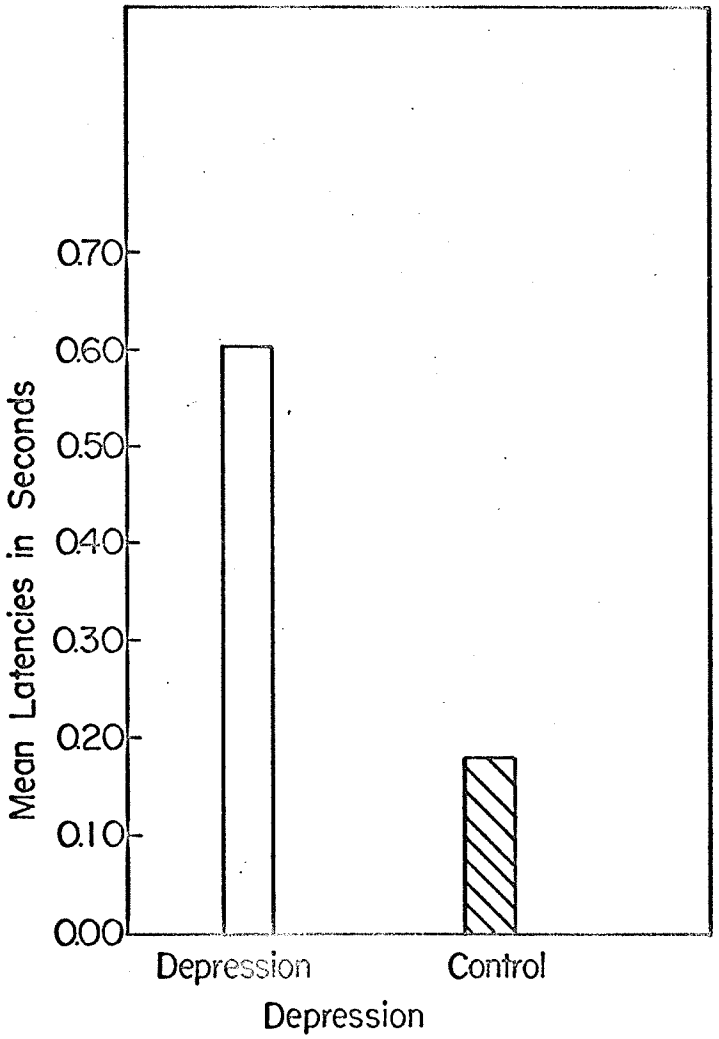

FIG. 1 MEAN ESCAPE LATENCIES OF DEPRESSED RATS AS COMPARED TO CONTROLS FOR TRIALS SIX TO CRITERION FOR COMBINED BARRIERS 1,2 & 3.

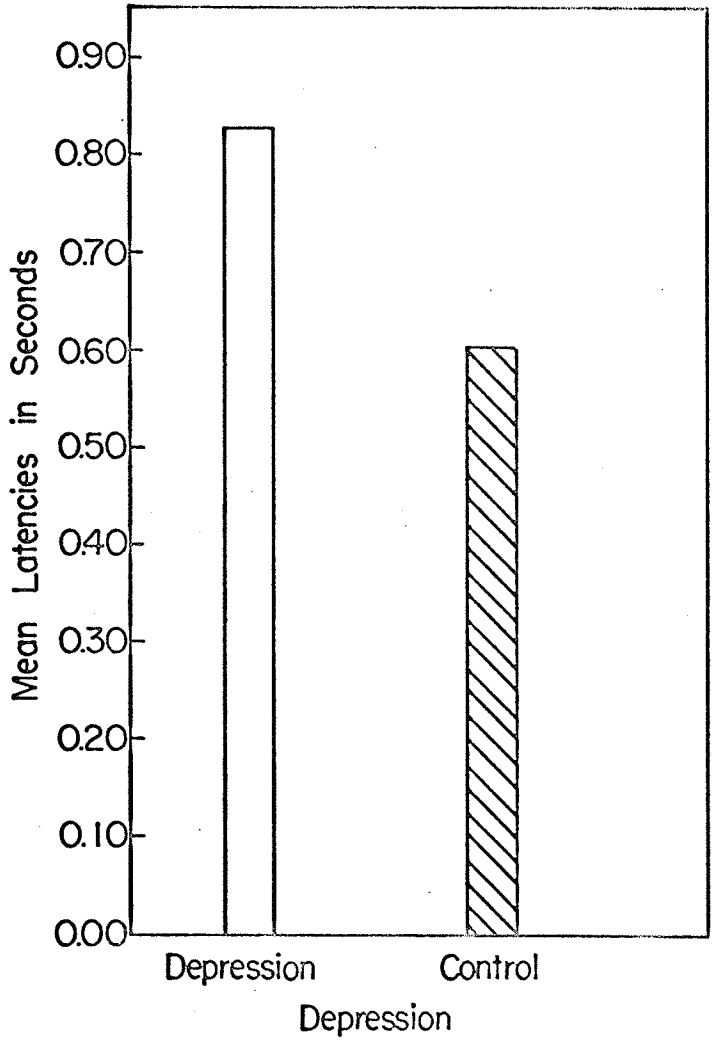

FIG. 2 MEAN ESCAPE LATENCIES OF DEPRESSED RATS AS COMPARED TO CONTROLS FOR COMBINED BARRIERS 4,2 & 5.

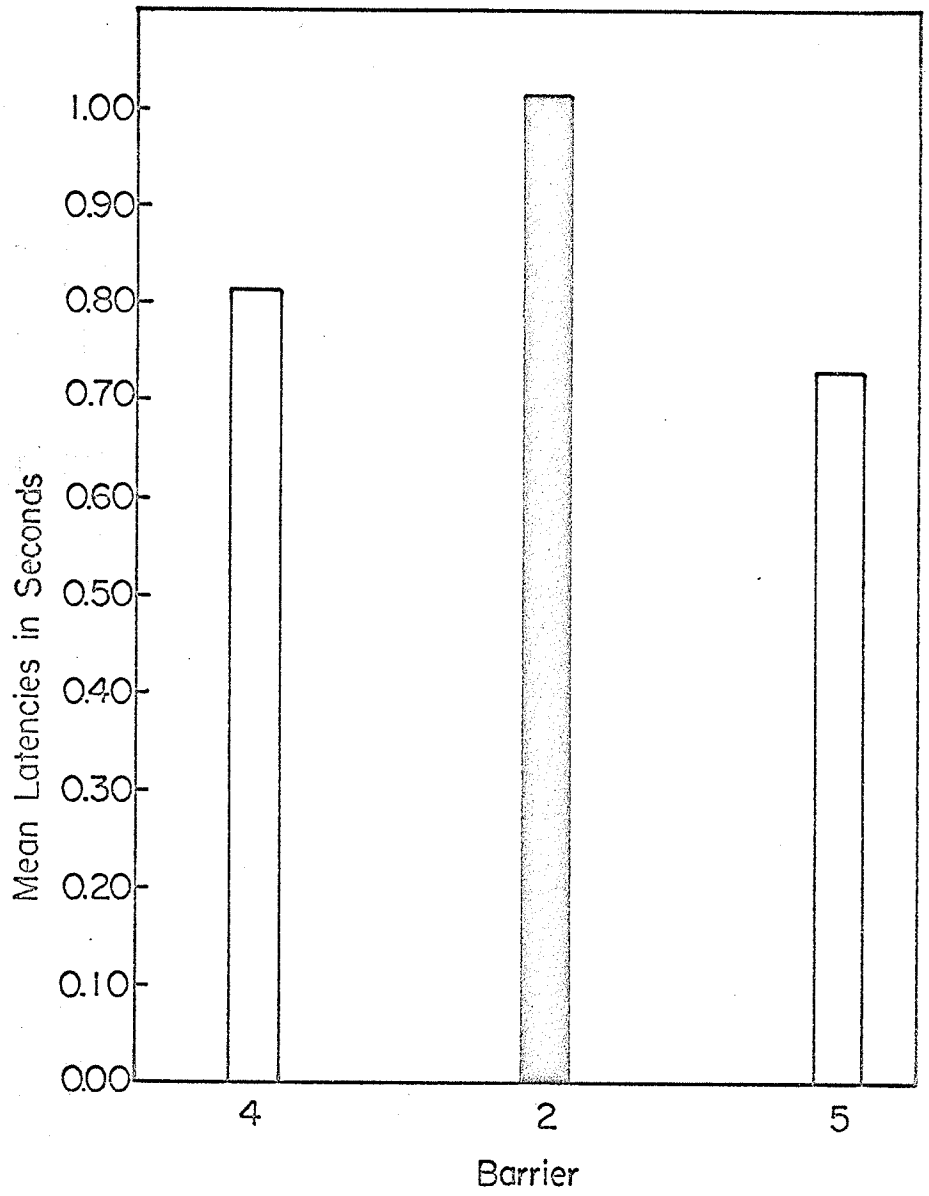

FIG.3 MEAN ESCAPE LATENCIES FOR FIRST FIVE TRIALS FOR COMBINED BARRIER OPENINGS WITH WIDTH VARIED.

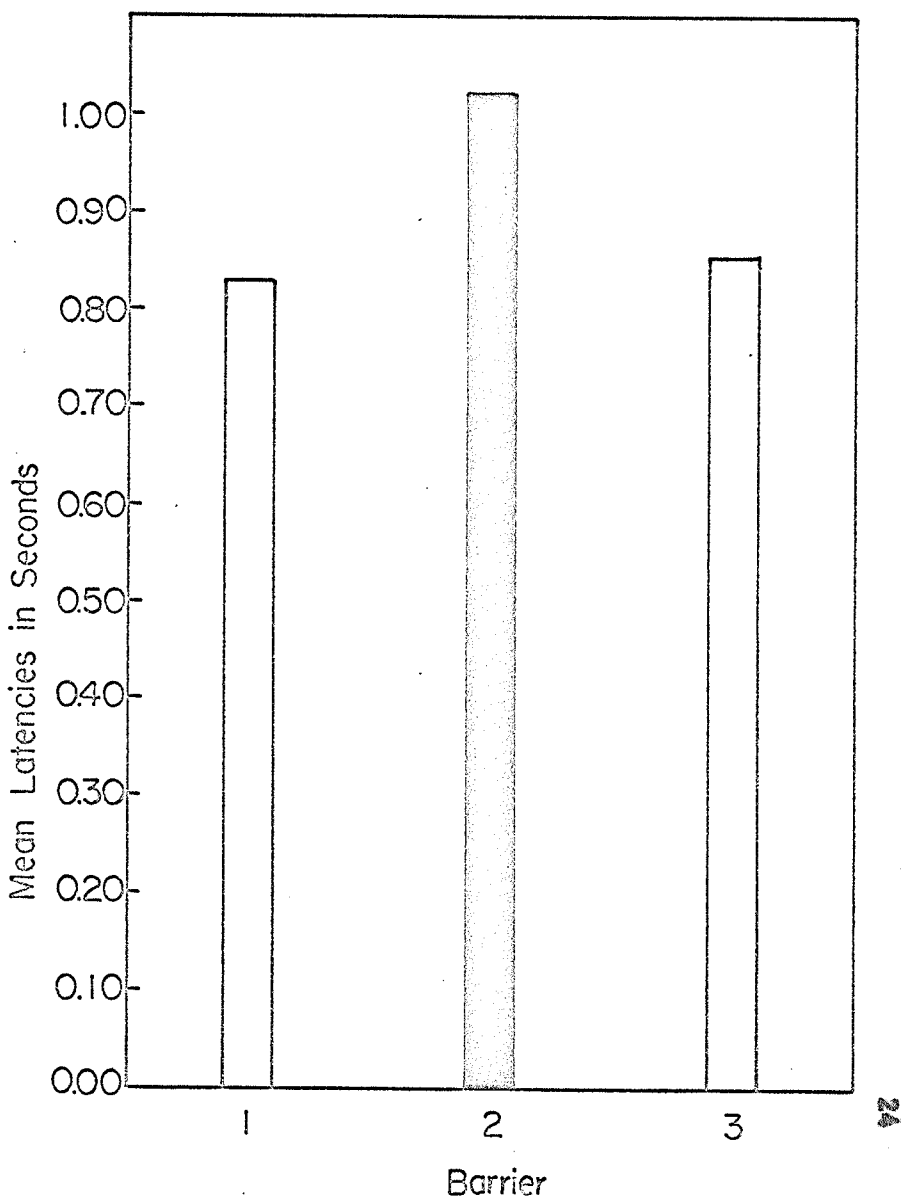

FIG. 4 MEAN ESCAPE LATENCIES FOR FIRST FIVE TRIALS FOR COMBINED BARRIER OPENINGS WITH HEIGHT VARIED.

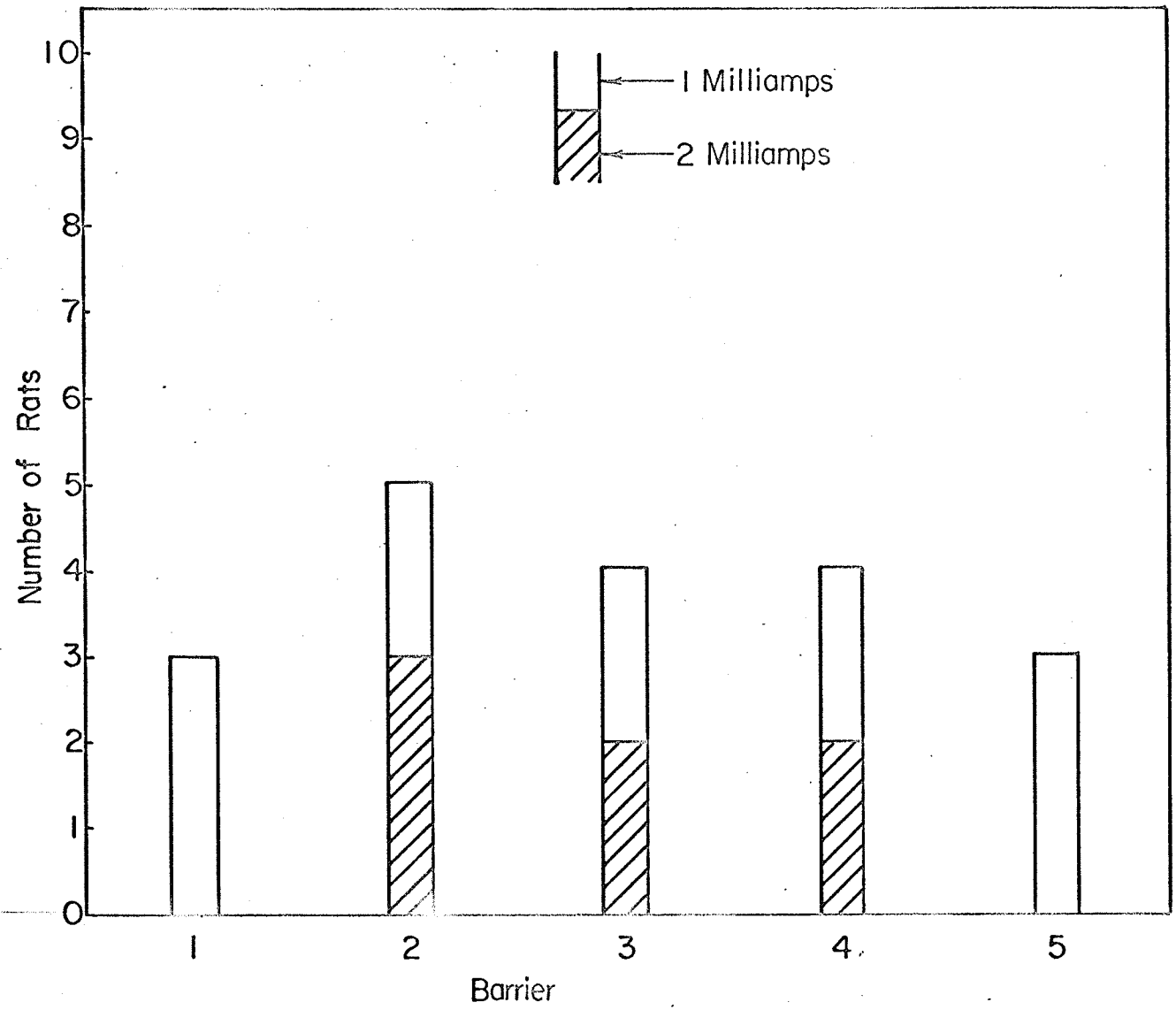

FIG. 5 NUMBER OF RATS FOR EACH BARRIER WHICH FAILED TO REACH CRITERION OF FIVE SUCCESSIVE AVOIDANCES.

to controls.

A significant barrier main effect was obtained from analysis of variance of mean escape latencies for combined barriers 1,2,3,4 (Tables 1,2,3). For combined barriers 4,2,5 analyses of variance of mean escape latencies to criterion (Table 1), of trials one to five (Fig. 3) and of trials six to criterion (Table 4) also yielded a significant barrier effect. A significant depression-barrier interaction is presented in Table 6. A Newman-Keuls test of the ordered differences of means for combined barriers 1,2,3 indicated that escape latencies to criterion for barrier 2 differed significantly from barriers 1 and 3, but escape latencies for barriers 1 and 3 did not differ significantly from each other ($p < .05$). Similarly, for combined barriers 4,2,5 a Newman-Keuls test showed that mean escape latencies to criterion for barrier 2 significantly differed from barriers 4 ($p < .05$) and 5 ($p < .01$). Also, a Newman-Keuls test revealed that mean escape latencies for trials one to five of barrier 4 significantly differed from those of barrier 5 and mean escape latencies for barrier 2 significantly differed from barriers 4 and 5 ($p < .01$); for trial six to criterion ordered differences of mean escape latencies for barrier 2 were significantly greater than those for barrier 5 ($p < .05$). A Newman-Keuls test for differences between ordered mean escape latencies for trials six to criterion for combined barriers 1,2,3,4,5 indicated that mean escape latencies for barrier 2 were significantly longer than for each of the other barriers. No other significant differences between barrier means were found ($p < .05$). From Fig. 5 it may be observed that number of rats unable to reach criterion of five consecutive avoidances is

identical for barriers 1 and 5. Also, for barriers 3 and 4 number of rats unable to acquire an avoidance response is the same. For shock intensity level of 2 ma., noticeably fewer rats failed to learn before the 75th trial. These results strongly suggest that acquisition seems poorest for barrier 2 for both control and depressed animals.

A significant drive main effect was obtained in analysis of mean escape latencies for trials six to criterion for combined barriers 1,2,3. (Fig. 6, Table 4). A significant depression-drive interaction for analysis of mean number of reinforcements is shown in Table 8. For combined barriers 4,2,5 analysis of variance of mean number of reinforcements yielded a significant drive main effect (Fig. 7). Significant depression-drive interactions for analyses of mean escape latencies for trials one to five and trials six to criterion are presented in Tables 5 and 2. From these results it appears that level of shock significantly influences rate of acquisition. Two-ma. USCD rats acquired an avoidance habit with significantly fewer reinforcements and lower mean escape latencies to criterion than 1-ma. USCD rats. Improvement in acquisition with increased shock intensity was not as evident in controls as in depressed rats.

A significant laterality main effect was revealed in analysis of mean escape latencies for trials six to criterion for combined barriers 1,2,3 (Fig. 8, Table 4). Significant depression-laterality interactions for analyses of mean escape latencies to criterion and for trials one to five are illustrated in Figures 9 and 10 (Tables 3 and 7). Side of cortex depressed produced no significant acquisition differences in analysis of combined barriers 4,2,5. From Figures 8, 9 and 10, it may

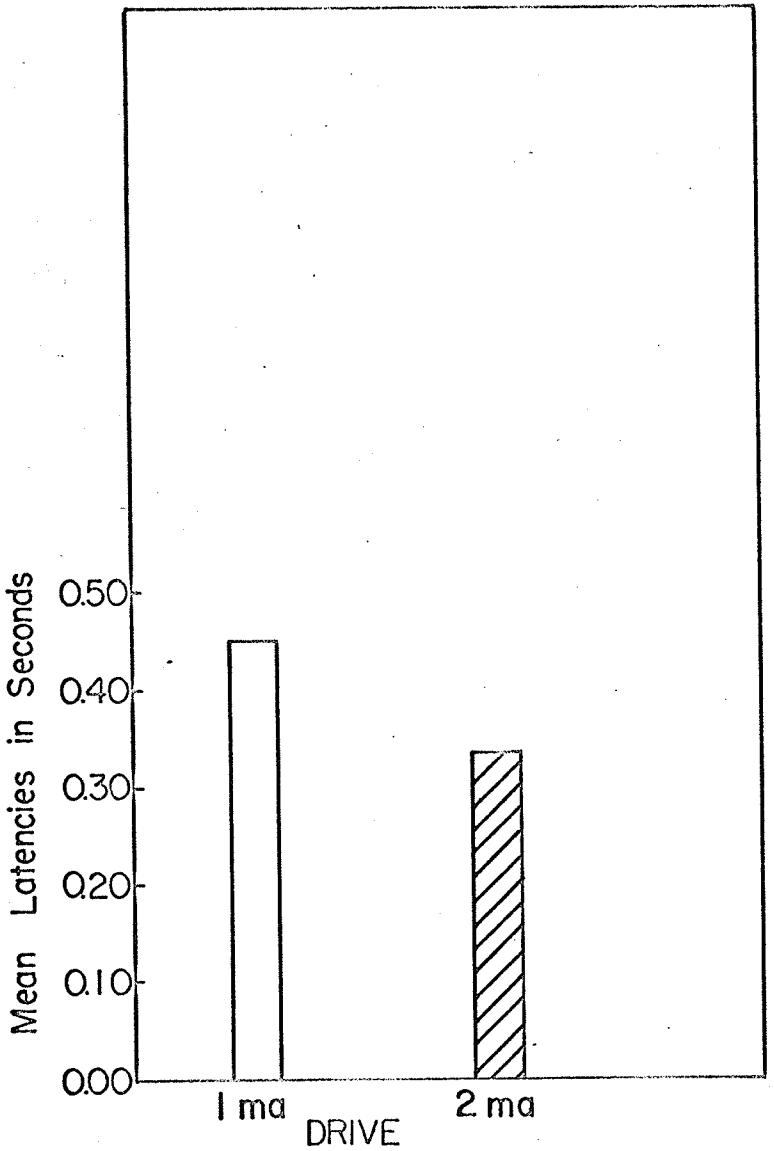

FIG. 6 MEAN ESCAPE LATENCIES AS A FUNCTION OF DRIVE LEVEL TO CRITERION FOR COMBINED BARRIERS 1, 2 & 3.

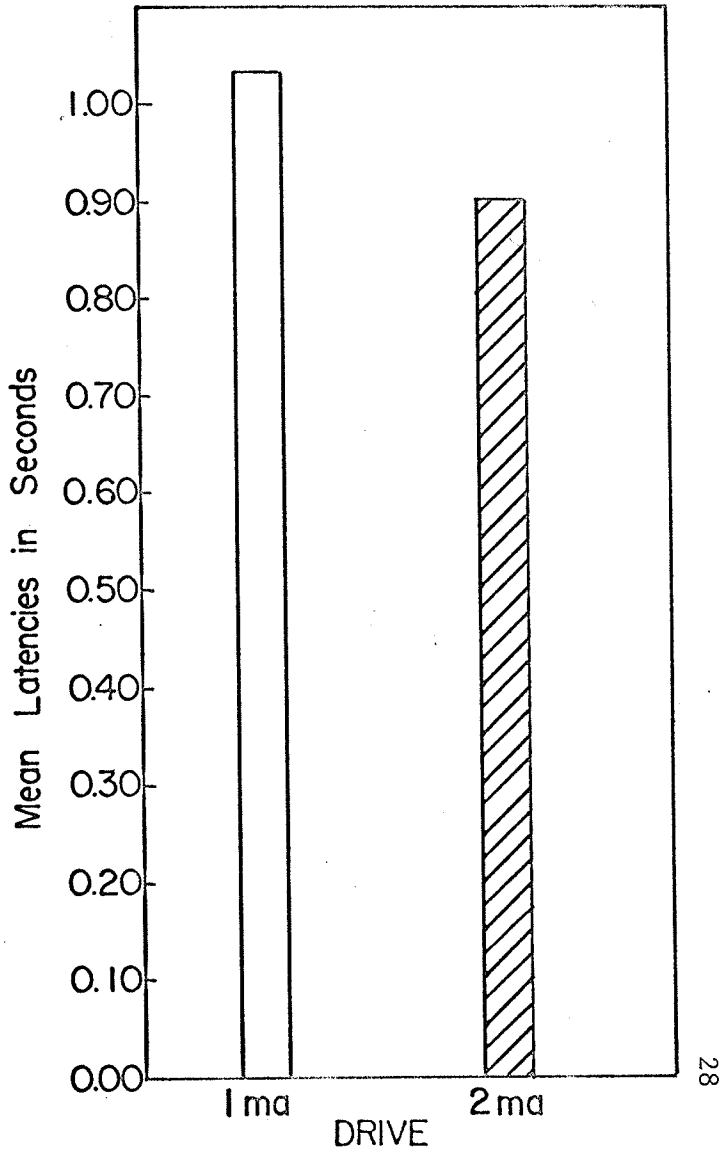

FIG. 7 MEAN NUMBER OF REINFORCEMENTS TO CRITERION AS A FUNCTION OF DRIVE LEVEL FOR COMBINED BARRIERS 4, 2 & 5.

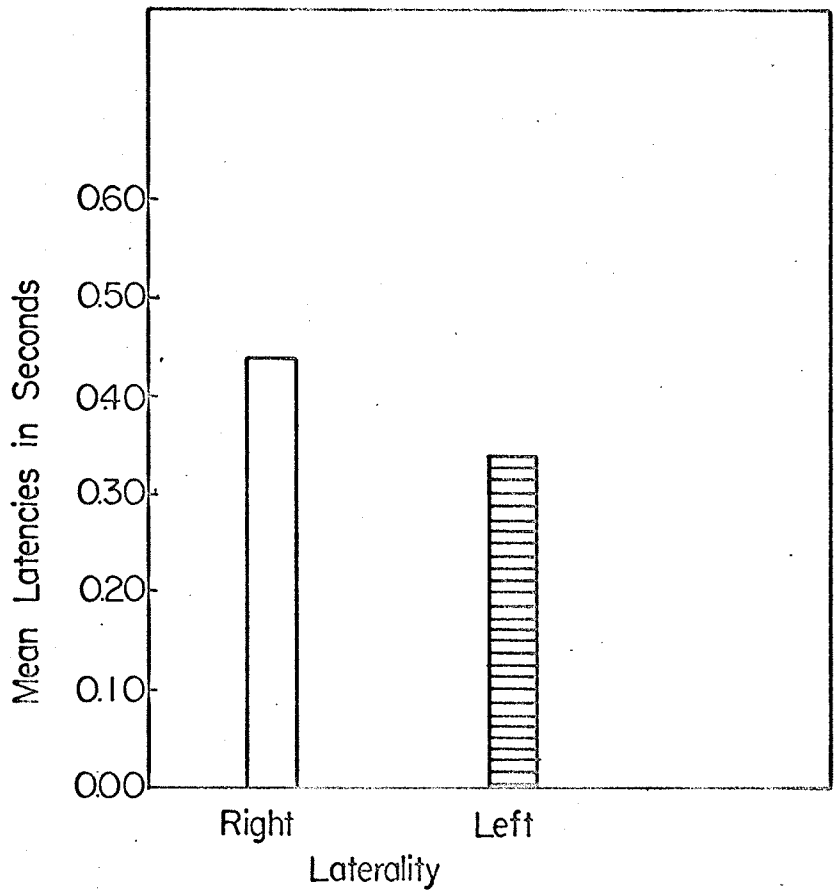

FIG. 8 MEAN ESCAPE LATENCIES FOR TRIALS SIX TO CRITERION AS A FUNCTION OF HEMISPHERE DEPRESSED FOR COMBINED BARRIERS 1, 2 & 3.

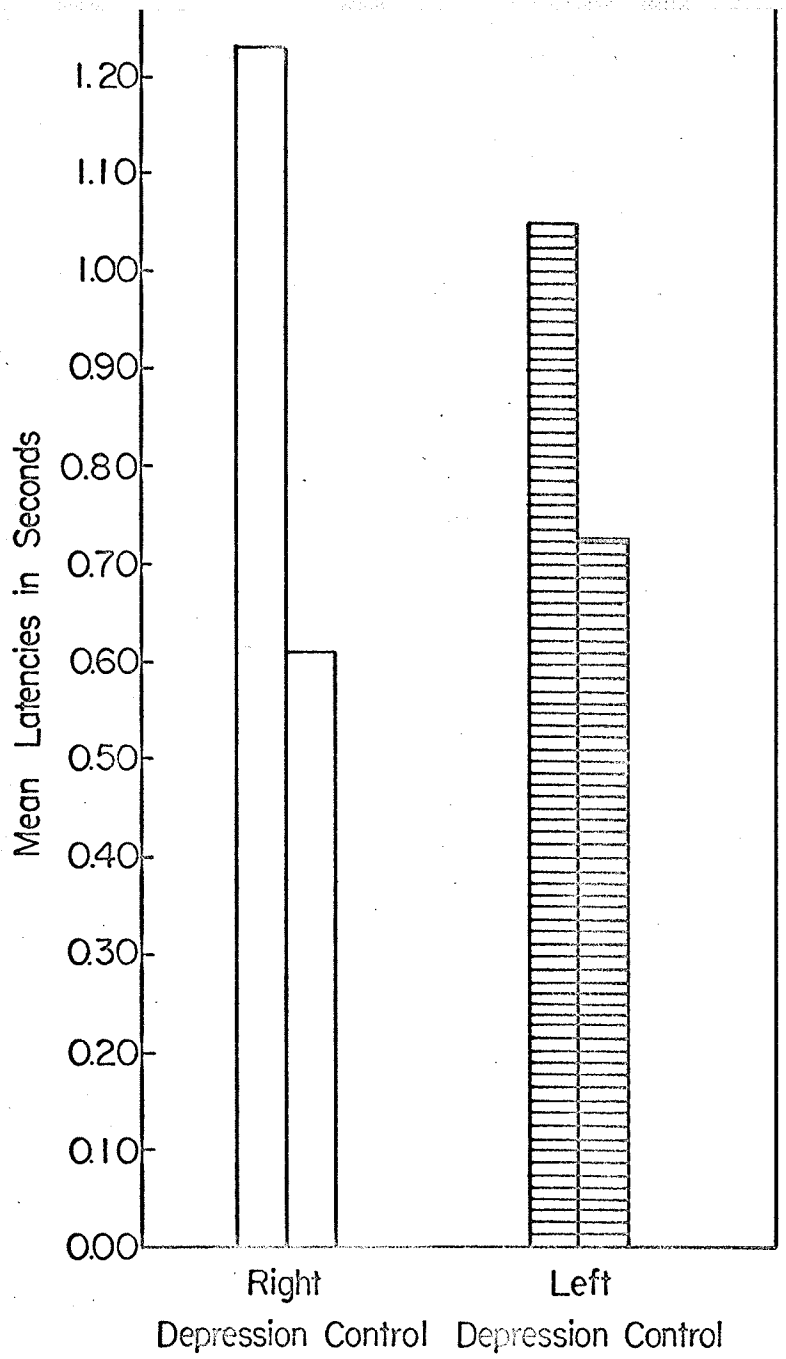

FIG. 9 MEAN ESCAPE LATENCIES FOR TRIALS ONE TO FIVE FOR COMBINED BARRIERS 1, 2 & 3 AS A FUNCTION OF DEPRESSION-LATERALITY INTERACTION

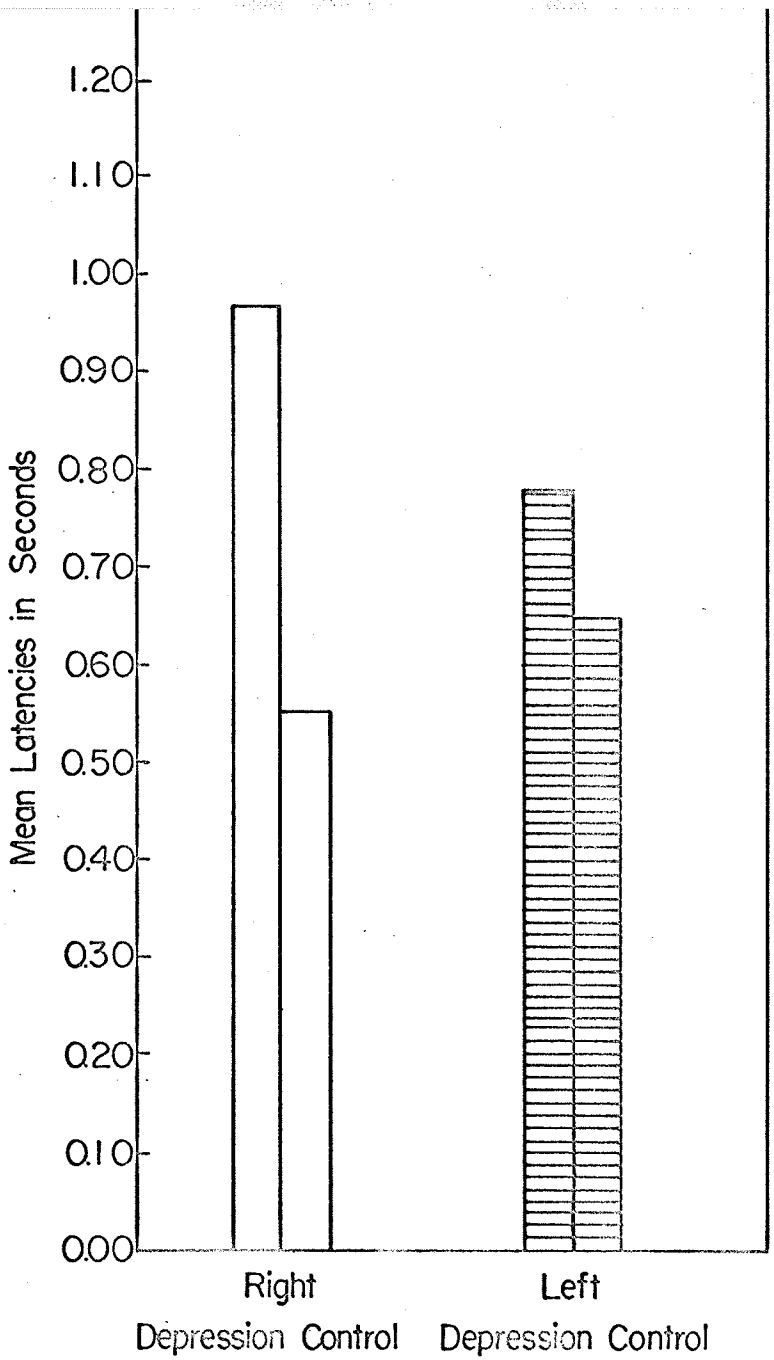

FIG. 10 MEAN ESCAPE LATENCIES TO CRITERION BARRIERS 1, 2 & 3 AS A FUNCTION OF DEPRESSION-LATERALITY INTERACTION.

be observed that one-way shuttle acquisition is more impaired by right hemispheric depression than by left hemispheric depression.

Statistical analysis of results indicated that acquisition deficits during USCD may be influenced by certain barrier dimensions, by shock intensity, and by laterality.

TABLE 1

ANALYSIS OF VARIANCE OF MEAN ESCAPE LATENCIES

TO CRITERION FOR COMBINED BARRIERS 4,2,5

Source of Variation	DF	SS	MS	F
Depression	1	1.5464	1.5464	26.05**
Barrier	2	0.7684	0.3842	6.47*
Depression x Barrier	2	0.0199	0.0099	0.17
Drive	1	0.1034	0.1034	1.74
Depression x Drive	1	0.1512	0.1512	2.55
Barrier x Drive	2	0.2132	0.1066	1.80
Depression x Barrier x Drive	2	0.2857	0.1428	2.41
Laterality	1	0.0111	0.111	0.19
Depression x Laterality	1	0.0568	0.0568	0.96
Barrier x Laterality	2	0.0285	0.0142	0.24
Depression x Barrier x Laterality	2	0.3070	0.1535	2.59
Drive x Laterality	1	0.0708	0.0708	1.19
Depression x Drive x Laterality	1	0.0149	0.0149	0.25
Barrier x Drive x Laterality	2	0.1076	0.0538	0.91
Depression x Barrier x Drive x Laterality	2	0.0398	0.0199	0.34
Within Cells	96	5.6977	0.0594	
Error Due to Approximation		0.1191		
Total	119	9.5416		

* $p < .005$

** $p < .001$

TABLE 2

ANALYSIS OF VARIANCE OF NUMBER OF REINFORCEMENTS IN TRIALS SIX TO CRITERION FOR COMBINED BARRIERS 4,2,5

Source of Variation	DF	SS	MS	F
Depression	1	3.6133	3.6133	43.94**
Barrier	2	0.5371	0.2685	3.27*
Depression x Barrier	2	0.1656	0.0828	1.01
Drive	1	0.0898	0.0898	1.09
Depression x Drive	1	0.4176	0.4176	5.08*
Barrier x Drive	2	0.1704	0.0852	1.04
Depression x Barrier x Drive	2	0.4920	0.2460	2.99
Laterality	1	0.0061	0.0061	0.07
Depression x Laterality	1	0.0707	0.0707	0.86
Barrier x Laterality	2	0.0371	0.0185	0.23
Depression x Barrier x Laterality	2	0.1926	0.0963	1.17
Drive x Laterality	1	0.0300	0.0300	0.36
Depression x Drive x Laterality	1	0.0168	0.0166	0.20
Barrier x Drive x Laterality	2	0.0544	0.0272	0.33
Depression x Barrier x Drive x Laterality	2	0.1273	0.0637	0.77
Within Cells	96	7.8946	0.0822	
Error Due to Approximation		0.0269		
Total	119	13.9420		

* $p < .05$

** $p < .001$

TABLE 3

ANALYSIS OF VARIANCE OF TRIALS ONE TO FIVE FOR

MEAN ESCAPE LATENCIES FOR COMBINED BARRIERS 1,2,3

Source of Variation	DF	SS	MS	F
Depression	1	6.8857	6.8857	68.55***
Barrier	2	0.9274	0.4637	4.62*
Depression x Barrier	2	0.0214	0.0107	0.11
Drive	1	0.2057	0.2057	2.05
Depression x Drive	1	0.0267	0.0267	0.27
Barrier x Drive	2	0.1766	0.0883	0.88
Depression x Barrier x Drive	2	0.0913	0.0457	0.45
Laterality	1	0.0356	0.0356	0.35
Depression x Laterality	1	0.7240	0.7240	7.21**
Barrier x Laterality	2	0.2427	0.1213	1.21
Depression x Barrier x Laterality	2	0.0469	0.0235	0.23
Drive x Laterality	1	0.0583	0.0583	0.58
Depression x Drive x Laterality	1	0.0012	0.0012	0.01
Barrier x Drive x Laterality	2	0.0335	0.0168	0.17
Depression x Barrier x Drive x Laterality	2	0.1506	0.0753	0.75
Within Cells	96	9.6438	0.1005	
Error Due to Approximation		-0.1518		
Total	119	19.1197		

* $p < .025$

** $p < .01$

*** $p < .001$

TABLE 4

ANALYSIS OF VARIANCE OF MEAN ESCAPE LATENCIES OF TRIALS SIX TO CRITERION FOR COMBINED BARRIERS 1,2,3

Source of Variation	DF	SS	MS	F
Depression	1	5.3541	5.3541	78.20**
Barrier	2	0.1946	0.0973	1.42
Depression x Barrier	2	0.0091	0.0045	0.07
Drive	1	0.3244	0.3244	4.74*
Depression x Drive	1	0.0557	0.0557	0.81
Barrier x Drive	2	0.0691	0.0345	0.50
Depression x Barrier x Drive	2	0.0220	0.0110	0.16
Laterality	1	0.3417	0.3417	4.99*
Depression x Laterality	1	0.0053	0.0053	0.08
Barrier x Laterality	2	0.2746	0.1373	2.01
Depression x Barrier x Laterality	2	0.1822	0.0911	1.33
Drive x Laterality	1	0.1113	0.1113	1.63
Depression x Drive x Laterality	1	0.0777	0.0777	1.14
Barrier x Drive x Laterality	2	0.0027	0.0014	0.02
Depression x Barrier x Drive x Laterality	2	0.2592	0.1296	1.89
Within Cells	96	6.5728	0.0685	
Error Due to Approximation		0.0267		
Total	119	13.8831		

* $p < .05$

** $p < .001$

TABLE 5

ANALYSIS OF VARIANCE OF MEAN ESCAPE LATENCIES IN TRIALS ONE TO FIVE FOR COMBINED BARRIERS 4,2,5

Source of Variance	DF	SS	MS	F
Depression	1	6.4794	6.1794	73.91***
Barrier	2	1.7901	0.8951	10.71***
Depression x Barrier	2	0.0275	0.0138	0.16
Drive	1	0.5395	0.5395	6.45*
Depression x Drive	1	0.7001	0.7001	8.37**
Barrier x Drive	2	0.3296	0.1648	1.97
Depression x Barrier x Drive	2	0.5012	0.2506	3.00
Laterality	1	0.0014	0.0014	0.02
Depression x Laterality	1	0.1939	0.1939	2.32
Barrier x Laterality	2	0.0476	0.0238	0.28
Depression x Barrier x Laterality	2	0.3258	0.1629	1.95
Drive x Laterality	1	0.0083	0.0083	0.10
Depression x Drive x Laterality	1	0.0068	0.0068	0.08
Barrier x Drive x Laterality	2	0.0199	0.0099	0.12
Depression x Barrier x Drive x Laterality	2	0.1852	0.0926	1.11
Within Cells	96	8.0264	0.0836	
Error Due to Approximation		-0.1667		
Total	119	18.7161		

* $p < .01$

** $p < .005$

*** $p < .001$

TABLE 6

ANALYSIS OF VARIANCE OF NUMBER OF REINFORCEMENTS TO CRITERION FOR COMBINED BARRIERS 4,2,5

Source of Variation	DF	SS	MS	F
Depression	1	7.5243	7.5243	84.62**
Barrier	2	0.5321	0.2660	2.99
Depression x Barrier	2	0.6681	0.3341	3.76*
Drive	1	1.5648	1.5648	17.60**
Depression x Drive	1	0.1019	0.1019	1.15
Barrier x Drive	2	0.4288	0.2144	2.41
Depression x Barrier x Drive	2	0.0311	0.0155	0.17
Laterality	1	0.0051	0.0051	0.06
Depression x Laterality	1	0.0507	0.0507	0.57
Barrier x Laterality	2	0.4534	0.2267	2.55
Depression x Barrier x Laterality	2	0.1884	0.0942	1.06
Drive x Laterality	1	0.0043	0.0043	0.05
Depression x Drive x Laterality	1	0.0250	0.0250	0.28
Barrier x Drive x Laterality	2	0.0670	0.0335	0.38
Depression x Barrier x Drive x Laterality	2	0.1519	0.0759	0.85
Within Cells	96	8.5363	0.0889	
Error Due to Approximation		-0.2010		
Total	119	20.1322		

* $p < .05$

** $p < .001$

TABLE 7

ANALYSIS OF VARIANCE OF MEAN ESCAPE LATENCIES TO CRITERION FOR COMBINED BARRIERS 1,2,3

Source of Variation	DF	SS	MS	F
Depression	1	2.1731	2.1739	29.26**
Barrier	2	0.4604	0.2302	3.10
Depression x Barrier	2	0.0962	0.0481	0.65
Drive	1	0.0020	0.0020	0.03
Depression x Drive	1	0.0378	0.0378	0.51
Barrier x Drive	2	0.1034	0.0517	0.70
Depression x Barrier x Drive	2	0.0329	0.0164	0.22
Laterality	1	0.0468	0.0468	0.63
Depression x Laterality	1	0.5297	0.5297	7.13*
Barrier x Laterality	2	0.0623	0.0311	0.42
Depression x Barrier x Laterality	2	0.0573	0.0286	0.39
Drive x Laterality	1	0.2716	0.2716	3.66
Depression x Drive x Laterality	1	0.0019	0.0019	0.03
Barrier x Drive x Laterality	2	0.0328	0.0164	0.22
Depression x Barrier x Drive x Laterality	2	0.0206	0.0103	0.14
Within Cells	96	7.1319	0.0743	
Error Due to Approximation		0.1079		
Total	119	11.1694		

* $p < .01$

** $p < .001$

TABLE 8

ANALYSIS OF VARIANCE OF NUMBER OF REINFORCEMENTS TO CRITERION FOR COMBINED BARRIERS 1,2,3

Source of Variation	DF	SS	MS	F
Depression	1	9.0237	9.0237	81.04**
Barrier	2	0.4517	0.2259	2.03
Depression x Barrier	2	0.5239	0.2620	2.38
Drive	1	0.5641	0.5641	5.07*
Depression x Drive	1	0.5088	0.5088	4.57*
Barrier x Drive	2	0.0277	0.0138	0.12
Depression x Barrier x Drive	2	0.1561	0.0780	0.70
Laterality	1	0.0118	0.0118	0.11
Depression x Laterality	1	0.0703	0.0703	0.63
Barrier x Laterality	2	0.2837	0.1418	1.27
Depression x Barrier x Laterality	2	0.3023	0.1512	1.36
Drive x Laterality	1	0.0748	0.0748	0.67
Depression x Drive x Laterality	1	0.0637	0.0637	0.57
Barrier x Drive x Laterality	2	0.0043	0.0022	0.02
Depression x Barrier x Drive x Laterality	2	0.3157	0.1578	1.42
Within Cells	96	10.6898	0.114	
Error Due to Approximation		-0.2035		
Total	119	22.8689		

* $p < .05$

** $p < .001$

DISCUSSION

Results of the present experiment indicate that the barrier parameters we employed to study stimulus, motor, and drive variables influence the rate of acquisition of a one-way shuttle.

Because rate of acquisition differed significantly only for barrier 2, vertical and horizontal parameters used failed to distinguish between the effects of SCD on motor debility and perceptual impairment. Therefore, our original assumptions about our measures were invalidated; barrier openings with width and height varied did not reliably evaluate differential effects of sensory and motor impairment. Properties unique to barrier 2, possibly a combination of stimulus and motor factors, may well have increased task difficulty in the general sense, retarding acquisition (Thompson, 1964; Thompson & Hjelle, 1965; Kunc & Kukleta, 1965). A single level of complexity, motor or sensory, may have been provided by barriers 1,3,4,5 and this may have been the reason no significant differences were obtained among them. Also, these results suggest that the extent of KCl-induced interference in performance may be task specific. An alternative explanation might be that barrier measures accurately reflected the underlying relationship between stimulus and motor effects, in line with the conception of pre- and post-central areas as one sensorimotor cortex (Mountcastle, 1968).

A major finding of the present experiment was the significant laterality effect obtained with barrier openings with height varied. For this barrier combination, performance of experimental rats significantly depended on hemisphere depressed. However, no significant

differences in rate of acquisition were obtained between the lowest and highest barriers. Complexity of the motor task may be indirectly reflected by laterality. But a motor explanation does not appear to entirely account for these findings. Equal numbers of rats failing to reach criterion of five successive avoidances on barriers 3 and 5 suggests motor complexity of the task may have been influenced by other factors, possibly sensory, in contributing to a significant laterality effect (Fig. 5). Markedly inferior rates of acquisition under right hemispheric depression supports the results of Kunc and Kukleta (1965). Hemispheric dominance, as demonstrated in handedness, did not appear to influence elaboration of a complex conditioned response (Buresova, Bures & Beran, 1958).

Our findings concerning shock intensities confirm results previously reported (Delprato, 1965; Delprato & Thompson, 1966; Thompson & Enter, 1967). Superior performance obtained with 2 ma. - USCD rats appears to be in line with a hypothesis of decreased threshold to shock suggested by Delprato and Thompson (1966). A related interpretation, decreased arousal, also seems to be consistent with our results. Heightened attention to the solution of the problem might be accompanied by an increase in orienting activity. Consequently, rate of acquisition would improve at the higher shock level. A suggestion of reduced "exploratory drive" under SCD, advanced by Delprato (1965) to account for reduced activity in an open field, conforms with this interpretation. The motivational effect of SCD seems to have interrelated components of decreased sensivity, arousal, and exploratory activity. Our data (Fig. 6,7) for both barrier

combinations tends to fit this composite view.

A decrease in spontaneous behavior was observed in treated animals in the present experiment. Although individual differences were found, depressed rats manifested a marked deficit in exploratory and locomotor behavior. Animals under depression showed aimless searching for the opening to the safe compartment. Their movements seemed to lack coordination. During acquisition, presence of depression could be detected by noting forepaws and hindpaw contralateral to the treated hemisphere limply extending through the grid floor. Similar observations were reported by Mogenson (1965). Depressed Ss seemed to experience more difficulty than controls in negotiating barrier openings, especially of the highest level (Barrier 3). Furthermore, this impairment in mobility is inseparable from kinesthetic deficits reported by Schneider (1967). Since rats were required to use the impaired limbs extensively, feedback from the impaired side was probably attenuated. Behavioral awkwardness observed in depressed rats might have contributed to longer escape latencies in experimental groups (Kunc & Kukleta, 1965; Freedman & Lash, 1966; Russell, Plotkin & Kleinman, 1968). Stimulus and motor complexity associated with widths and heights of barrier openings were well within the capability of the majority of USCD and all control animals (Fig. 5).

Emotionality did not appear to contribute to observed changes in performance. There seemed to be no differences in number of vocalizations between experimental and control groups. Tapp (1962), Delprato (1965), and Carlson (1967) obtained similar findings.

Results of the present experiment suggest that disruption of behavior by spreading cortical depression was attenuated at higher shock levels. Furthermore, laterality was a significant effect only with the set of barrier openings with height varied. Finally, the relative roles of sensory and motor deficits in performance changes were not conclusively determined by the one-way shuttle method employed.

SUMMARY

The present experiment investigated the influence of unilateral spreading cortical depression on motor, stimulus, and drive variables in a one-way shuttle setting.

Acquisition of an aversively-reinforced response in unilaterally depressed rats (n = 100, 10 Ss per group) was compared to saline controls (n = 100, 10 Ss per group) in two 2 x 3 x 2 x 2 factorial designs. Height of barrier opening was varied in one design and width of barrier opening was varied in the other. Effects of shock intensity and laterality were also studied. Rate of acquisition was measured by mean escape latencies and by number of reinforcements to criterion of five successive avoidances or seventy-five trials.

The relative roles of motor and sensory disturbances in performance changes were not decisively established by the barrier dimensions employed. Disruption of behavior was enhanced in 1 ma.-depressed animals as compared to 2 ma.-depressed animals. Finally, a major finding was that acquisition under right hemispheric depression was markedly inferior as compared to left hemispheric depression.

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APPENDIX A

MEAN ESCAPE LATENCIES TO CRITERION

Barrier 1

1 MA

Rat No.	SDL	No. of Reinf.	SDR	No. of Reinf.	NaCl	No. of Reinf.	Order	Rat No.	Normal	No. of Reinf.
100	8.285	2	7.285	8	10.40	1	R-L	95	2.786	5
101	14.806	25	27.956	7	1.39	1	R-L	96	1.470	3
102	5.170	70	4.684	32	23.783	14	R-L	97	2.330	6
103	32.538	20	6.648	75	2.26	1	R-L	151	4.907	6
104	3.831	7	2.41	4	1.616	10	R-L	150	2.245	4
105	2.872	23	5.421	11	4.270	2	L-R	114	1.550	5
106	23.647	6	2.675	4	1.893	3	L-R	121	2.522	5
107	5.439	8	15.957	7	1.61	1	L-R	122	3.462	5
129	3.355	75	3.020	73	2.353	6	L-R	123	2.864	5
109	3.369	31	18.690	2	2.670	7	L-R	124	3.126	5
$\bar{X}$	10.331	26.70	9.474	22.30	5.224	4.60			2.776	4.90
S^2	104.51	673.788	73.199	813.789	447.46	20.71			0.873	0.767

2 MA

130	5.328	14	4.517	3	3.590	1	L-R	125	2.301	6
131	4.355	8	3.034	41	1.420	1	L-R	126	6.370	2
132	3.977	14	5.235	2	0.000	0	L-R	127	1.757	3
133	4.595	4	11.213	12	0.000	0	L-R	128	2.350	4
134	3.918	5	4.770	6	1.400	2	L-R	143	3.500	7
135	5.818	6	16.276	20	1.898	6	R-L	142	4.335	6
136	15.122	5	6.794	13	1.080	1	R-L	141	2.603	3
137	1.807	7	5.827	6	2.800	1	R-L	140	2.334	5
138	8.625	2	9.273	6	2.160	1	R-L	144	1.390	5
139	3.254	22	8.887	9	0.917	10	R-L	145	2.800	3
$\bar{X}$	5.679	8.70	7.582	11.8	1.527	2.300			2.973	4.400
S^2	14.192	37.57	15.623	133.73	1.297	10.233			2.118	2.711

MEAN ESCAPE LATENCIES TO CRITERION

Barrier 2

1 MA

Rat No.	SDL	No. of Reinf.		No. of Reinf.	NaCl	No. of Reinf.	Order	Rat No.	Normal	No. of Reinf.
44	3.798	75	2.880	74	2.670	7	L-R	22	2.110	4
66	13.170	75	4.171	75	3.298	75	L-R	27	2.628	6
67	5.311	75	4.295	75	1.957	42	L-R	28	4.890	2
68	5.348	7	5.431	19	1.645	2	L-R	29	1.450	6
69	5.880	17	13.188	25	2.777	4	R-L	30	2.477	7
70	13.060	11	5.671	74	3.007	3	R-L	31	1.847	6
75	27.566	5	10.407	19	17.83	1	R-L	32	1.831	8
74	22.380	2	3.292	21	21.36	1	R-L	25	38.510	1
76	3.328	70	4.181	7	1.007	3	L-R	46	2.441	9
78	6.643	10	7.043	9	2.274	56	L-R	62	20.697	6
$\bar{X}$	10.649	34.7	6.055	39.800	5.783	19.4			7.889	5.5
S^2	70.220	1146.900	11.032	919.95	54.138	761.155			149.49	6.278
				2 MA						
79	10.127	8	10.492	4	1.490	1	R-L	63	2.987	4
80	2.392	73	3.236	75	1.538	20	R-L	71	1.103	7
81	4.389	20	6.437	9	1.055	4	L-R	73	1.947	4
86	6.779	18	21.116	7	4.300	2	L-R	72	11.266	5
85	12.083	7	17.212	5	22.81	2	R-L	82	14.043	3
88	33.392	10	51.365	18	11.285	2	R-L	83	3.610	2
89	4.446	74	3.490	8	2.13	1	L-R	87	6.847	3
90	6.740	65	4.649	74	1.586	5	L-R	84	2.268	7
91	16.034	9	10.518	10	1.28	1	R-L	93	3.745	4
92	6.267	3	7.244	11	0.942	4	R-L	94	7.665	2
$\bar{X}$	10.26	28.700	13.576	22.100	4.841	4.100			5.58	4.100
S^2	82.467	868.900	210.656	777.433	49.661	33.433			18.673	3.211

MEAN ESCAPE LATENCIES TO CRITERION

Barrier 3

1 MA

Rat No.	SDL	No. of Reinf.	SDR	No. of Reinf.	NaCl	No. of Reinf.	Order	Rat No.	Normal	No. of Reinf.
146	5.225	13	9.447	4	1.811	6	L-R	98	1.955	17
147	30.976	11	5.185	4	4.62	1	L-R	99	2.476	18
148	3.174	65	35.347	3	2.050	2	L-R		1.575	12
149	2.692	75	6.612	75	1.510	4	L-R	160	2.181	8
110	3.500	9	3.200	7	1.261	20	L-R	161	1.887	6
111	3.203	24	6.704	75	2.019	19	R-L	116	3.198	5
152	12.786	10	7.320	8	1.200	3	R-L	117	1.686	11
153	3.953	17	2.565	36	1.235	6	R-L	118	38.450	4
158	19.245	8	24.226	34	1.825	9	R-L	119	1.446	30
159	3.124	7	2.086	73	1.227	4	R-L	120	2.041	7
$\bar{X}$	8.786	23.900	10.269	31.900	1.875	7.400			5.690	11.800
s^2	90.437	620.766	117.962	100.98	1.043	45.82			132.748	63.955

2 MA

162	2.329	75	4.094	75	2.526	16	R-L	225	2.073	4
163	5.705	15	8.165	6	1.695	4	R-L	224	1.118	10
164	4.621	10	3.100	10	1.073	3	R-L	222	1.935	2
165	5.113	3	35.959	19	1.818	5	R-L	221	1.158	5
207	3.222	12	8.426	5	2.400	1	R-L	213	1.333	6
208	3.572	6	6.183	18	5.105	4	L-R	113	3.95	1
209	3.165	17	3.114	5	1.577	3	L-R	112	1.020	5
210	3.690	4	2.605	6	1.337	3	L-R	155	1.363	4
211	4.554	11	3.619	74	1.905	2	L-R	156	1.933	3
212	4.978	5	2.768	6	2.540	1	L-R	154	1.825	2
$\bar{X}$	4.093	15.800	7.802	22.400	2.197	4.200			1.769	4.200
s^2	1.117	454.844	102.644	780.71	1.285	18.840			0.735	6.622

MEAN ESCAPE LATENCIES TO CRITERION

Barrier 4

1 MA

Rat No.	SDL	No. of Reinf.	SDR	No. of Reinf.	NaCl	No. of Reinf.	Order	Rat No.	Normal	No. of
214	4.934	45	7.548	9	0.902	4	L-R	219	3.513	6
215	23.864	75	3.549	29	2.643	15	L-R	223	4.670	2
216	6.327	4	2.802	40	1.190	4	L-R	227	3.084	5
226	7.362	4	6.210	1	1.670	1	L-R	235	1.657	11
218	2.339	74	4.337	7	1.326	12	L-R	228	1.374	8
254	15.387	3	8.190	40	3.190	2	R-L	244	5.426	7
230	7.216	7	4.570	19	1.867	3	R-L	245	1.377	4
231	6.771	9	12.933	6	1.393	6	R-L	255	3.880	3
233	6.287	8	4.126	75	3.211	10	R-L	258	3.250	3
232	5.158	31	2.497	60	2.633	3	R-L	259	1.780	3
$\bar{X}$	8.565	26.000	5.667	28.600	2.002	6.000			3.001	5.200
S^2	40.081	840.222	10.239	623.822	0.722	22.222			2.040	7.955

2 MA

236	5.127	4	3.824	13	3.055	2	R-L	229	2.206	5
256	3.194	67	3.322	71	1.108	13	R-L	251	2.185	2
238	3.343	23	8.224	7	1.420	3	R-L	242	1.550	6
239	2.276	14	1.875	8	1.970	1	R-L	243	1.404	5
250	3.086	7	6.355	57	2.095	2	R-L	247	2.747	4
246	3.281	12	4.900	4	1.150	1	L-R	240	9.066	13
248	3.422	9	9.673	3	3.900	2	L-R	241	1.319	11
249	12.323	3	13.900	2	12.780	1	L-R	253	1.625	2
252	5.002	5	7.035	6	1.590	1	L-R	260	4.432	9
257	2.179	72	3.259	34	1.510	6	L-R	261	2.005	2
$\bar{X}$	4.323	21.600	6.234	20.50	3.057	3.200			2.853	5.900
S^2	8.833	672.93	13.238	621.166	12.446	14.177			5.602	15.211

MEAN ESCAPE LATENCIES TO CRITERION

Barrier 5

1 MA

Rat No.	SDL	No. of Reinf.	SDR	No. of Reinf.	NaCl	No. of Reinf.	Order	Rat No.	Normal	No. of Reinf.
170	40.242	75	9.447	6	1.405	2	L-R	166	1.619	22
171	4.332	12	3.330	4	1.657	3	L-R	167	2.917	3
172	9.084	10	3.538	19	3.062	41	L-R	168	1.870	3
173	9.609	16	3.017	8	1.880	1	L-R	169	2.740	4
174	3.811	8	2.615	6	1.822	4	L-R	179	2.637	3
175	2.700	6	19.507	18	1.277	3	R-L	186	2.917	3
176	6.648	5	2.707	7	2.633	3	R-L	187	2.220	2
177	2.523	20	7.950	16	2.378	22	R-L	197	1.292	4
181	1.900	8	4.565	73	1.728	5	R-L	205	1.422	5
178	2.865	11	3.954	61	2.905	2	R-L	206	1.212	5
$\bar{X}$	8.37	17.100	6.063	21.800	2.074	8.600			2.085	5.4
S^2	132.93	434.544	31.660	604.40	0.393	166.933			0.472	34.933
				2 MA						
182	2.095	2	3.397	4	1.55	1	R-L	184	1.327	4
188	10.057	4	3.352	6	4.500	2	R-L	185	3.082	6
189	1.928	5	3.136	10	2.120	3	R-L	183	4.043	19
190	1.723	3	2.655	6	1.335	2	R-L	198	2.608	7
191	1.542	6	1.988	5	1.770	1	R-L	199	2.063	3
192	3.263	3	3.023	6	7.620	1	L-R	200	3.715	2
193	3.014	12	2.112	4	0.957	20	L-R	201	1.510	5
194	1.531	40	10.26	1	2.150	3	L-R	202	1.793	6
195	3.745	11	4.050	2	1.297	3	L-R	203	3.070	3
196	2.977	4	6.524	19	2.900	4	L-R	204	2.070	3
$\bar{X}$	3.185	9.000	4.049	6.300	2.622	4.000			2.527	5.800
S^2	6.431	130.000	6.382	26.011	4.120	32.666			0.860	24.177

APPENDIX B

MEAN ESCAPE LATENCIES FOR TRIALS 1-5

Barrier 1

1 MA

Rat No.	SDL	No. of Reinf.	SDR	No. of Reinf.	NaCl	No. of Reinf.	Order	Rat No.	Control	No. of Reinf.
100	8.28	2	8.28	4	10.40	1	R-L	95	3.75	3
101	60.00	5	38.16	5	1.39	1	R-L	96	1.97	3
102	18.35	5	17.42	5	262.80	5	R-L	97	2.96	3
103	60.00	5	60.00	5	2.26	1	R-L	151	5.56	5
104	30.86	5	2.41	4	6.17	3	R-L	150	2.63	3
105	6.41	5	9.42	5	3.46	1	L-R	114	1.55	5
106	27.92	5	3.38	2	5.68	3	L-R	121	2.52	5
107	6.75	5	21.20	5	1.61	1	L-R	122	3.46	5
129	15.45	5	13.84	5	9.51	4	L-R	103	2.86	5
109	4.08	5	18.69	2	11.25	4	L-R	104	3.75	4
					2 MA					
130	5.95	5	4.52	3	3.59	1	L-R	125	2.59	5
131	5.95	5	9.78	5	1.42	1	L-R	126	6.37	2
132	9.42	5	5.24	2	0	0	L-R	127	1.76	3
133	4.60	4	15.10	5	0	0	L-R	128	2.35	4
134	3.92	5	2.41	4	2.80	2	L-R	143	6.99	3
135	7.29	4	46.07	5	6.60	4	R-L	142	8.39	5
136	23.61	2	8.39	5	1.08	1	R-L	141	2.60	3
137	8.96	4	5.70	5	2.80	1	R-L	140	2.33	5
138	8.63	2	11.57	4	2.16	1	R-L	144	1.59	4
139	9.61	5	13.75	5	3.79	4	R-L	145	2.80	3

MEAN ESCAPE LATENCIES FOR TRIALS 1-5

Barrier 2

1 MA

Rat No.	SDL	No. of Reinf.	SDR	No. of Reinf.	NaCl	No. of Reinf.	Order	Rat No.	Control	No. of Reinf.
44	14.62	5	10.15	5	13.42	5	L-R	22	2.11	4
66	50.79	5	16.44	5	18.70	5	L-R	27	2.96	4
67	29.35	5	12.86	5	17.57	5	L-R	28	4.89	2
68	5.29	5	8.29	5	3.29	2	L-R	29	1.97	3
69	5.32	5	32.15	5	11.11	4	R-L	30	.48	5
70	23.27	5	41.13	5	5.28	1	R-L	31	2.06	5
75	40.66	3	32.15	5	17.83	1	R-L	32	2.48	5
74	22.38	2	9.27	5	21.36	1	R-L	25	38.51	1
76	7.34	5	8.05	3	3.02	3	L-R	46	3.66	4
78	9.08	5	9.54	5	27.39	5	L-R	62	29.43	4
					2 MA					
79	13.94	4	10.49	4	1.49	1	R-L	63	2.33	3
80	6.26	5	10.19	5	9.88	5	R-L	71	1.23	4
81	6.90	5	8.50	5	4.22	4	L-R	78	2.31	3
86	11.35	5	26.41	5	8.60	2	L-R	72	11.27	5
85	15.58	5	17.21	5	22.81	1	R-L	82	14.21	3
88	55.50	5	60.00	5	22.57	2	R-L	83	3.61	2
89	18.70	5	3.98	5	2.13	1	L-R	87	9.03	2
90	43.04	5	21.06	5	6.90	4	L-R	84	4.85	2
91	19.02	5	17.47	5	1.28	1	R-L	93	3.75	4
92	6.29	3	15.35	4	2.92	3	R-L	94	17.67	2

MEAN ESCAPE LATENCIES FOR TRIALS 1-5

Barrier 3

1 MA

Rat No.	SDL	No. of Reinf.	SDR	No. of Reinf.	NaCl	No. of Reinf.	Order	Rat No.	Control	No. of Reinf.
146	10.19	5	11.99	3	8.77	4	L-R	98	2.89	5
147	60.00	5	5.19	4	4.62	1	L-R	99	4.92	5
148	13.91	5	35.34	3	4.10	2	L-R		2.48	5
149	9.90	5	36.50	5	6.04	4	L-R	160	3.05	5
110	4.92	5	3.81	5	8.52	3	L-R	161	2.96	3
111	5.13	5	32.07	5	13.64	5	R-L	116	5.86	2
152	24.36	4	10.81	5	3.60	3	R-L	117	2.51	4
153	7.59	5	2.90	5	5.34	4	R-L	118	38.45	4
158	28.48	5	57.71	5	8.87	3	R-L	119	2.43	4
159	3.87	5	8.44	5	2.54	2	R-L	120	2.34	5
					2 MA					
162	5.65	5	22.74	5	12.96	5	R-L	154	1.83	2
163	10.51	5	9.40	5	4.84	2	R-L	225	2.07	4
164	7.91	5	4.35	5	2.10	2	R-L	224	1.39	5
165	5.11	3	60.00	5	7.41	3	R-L	222	1.94	2
207	4.64	5	9.89	4	2.40	1	R-L	221	1.16	5
208	3.37	5	16.89	5	19.74	3	L-R	213	1.42	5
209	16.34	2	3.79	4	4.73	3	L-R	113	13.95	1
210	3.69	4	2.81	5	3.13	2	L-R	112	1.02	5
211	8.54	5	5.02	5	3.81	2	L-R	155	1.36	4
212	5.85	4	3.54	4	2.54	1	L-R	156	1.93	3

MEAN ESCAPE LATENCIES FOR TRIALS 1-5

Barrier 4

1 MA

Rat No.	SDL	No. of Reinf.	SDR	No. of Reinf.	NaCl	No. of Reinf.	Order	Rat No.	Control	No. of Reinf.
214	24.84	5	15.17	4	3.61	4	L-R	219	4.05	5
215	60.00	4	8.73	5	23.58	5	L-R	223	4.67	2
216	6.33	4	7.71	5	4.76	4	L-R	227	3.70	4
226	7.36	4	6.21	1	1.67	1	L-R	235	3.17	4
218	4.69	5	4.84	5	6.08	4	L-R	228	1.67	5
254	15.39	3	23.39	5	6.38	2	R-L	244	8.55	4
230	8.06	5	7.63	5	5.60	3	R-L	245	1.38	4
231	5.69	5	15.30	5	4.47	4	R-L	255	3.88	3
233	7.92	5	31.75	5	7.93	4	R-L	258	3.25	3
232	19.93	5	5.46	5	7.90	3	R-L	259	1.78	3

2 MA

236	6.06	3	6.32	5	6.11	2	R-L	229	2.31	4
256	1.74	5	20.94	5	7.24	5	R-L	251	2.19	2
238	6.23	5	9.94	5	4.26	3	R-L	242	1.71	5
239	4.34	5	2.24	5	1.27	1	R-L	243	1.40	4
250	2.93	5	26.19	5	4.19	2	R-L	247	2.75	4
246	3.70	5	4.90	4	1.15	1	L-R	240	15.09	5
248	4.76	5	9.67	3	7.90	2	L-R	241	1.92	5
249	12,32	3	13.90	2	12.78	1	L-R	253	1.63	2
252	6.03	4	4.48	3	1.59	1	L-R	260	4.98	3
257	7.43	5	10.49	5	7.58	4	L-R	261	2.00	2

MEAN ESCAPE LATENCIES FOR TRIALS 1-5

BARRIER 5

1 MA

Rat No.	SDL	No. of Reinf.	SDR	No. of Reinf.	NaCl	No. of Reinf.	Order	Rat No.	Control	No. of Reinf.
170	58.20	5	10.34	5	2.81	2	L-R	166	2.67	4
171	7.00	5	3.33	4	2.16	1	L-R	167	2.92	3
172	15.49	5	6.45	5	17.16	5	L-R	168	1.88	3
173	25.78	5	3.22	5	1.88	1	L-R	169	2.74	4
174	4.78	5	3.06	5	4.46	2	L-R	179	2.64	3
175	2.95	4	60.00	5	2.74	2	R-L	196	2.92	3
176	7.98	4	3.25	5	7.90	3	R-L	187	2.22	2
177	3.40	5	25.13	4	17.19	5	R-L	197	1.29	4
181	2.46	5	13.95	4	6.18	3	R-L	205	1.42	5
178	4.50	5	22.20	5	5.81	2	R-L	206	1.21	5

2 MA

182	2.09	2	3.40	4	1.55	1	R-L	184	1.33	4
188	14.83	2	3.87	5	9.00	2	R-L	185	3.58	5
189	1.93	5	2.86	5	6.36	3	R-L	183	11.13	5
190	1.72	3	3.02	5	2.20	2	R-L	198	1.42	5
191	1.79	3	1.99	5	1.79	1	R-L	199	2.06	3
192	3.26	3	2.00	5	7.62	1	L-R	200	3.72	2
193	4.85	5	2.11	4	5.95	4	L-R	201	1.62	4
194	3.46	5	10.26	1	6.45	3	L-R	202	1.99	5
195	5.65	5	4.05	2	3.89	3	L-R	203	3.07	3
196	2.98	4	8.83	5	10.20	2	L-R	204	2.07	3

APPENDIX C

$\bar{X}$ ESCAPE LATENCIES FOR TRIALS 6-75

Barrier 1

1 MA

Rat No.	SDL	SDR	Order	Rat No.	Control
100	0.00	6.29	R-L	95	1.34
101	3.51	2.44	R-L	96	0.00
102	4.16	2.33	R-L	97	1.70
103	23.78	4.98	R-L	151	1.63
104	3.75	0.00	R-L	150	1.08
105	1.89	2.09	L-R	114	0.00
106	2.27	1.97	L-R	121	0.00
107	3.26	2.85	L-R	122	0.00
108	2.49	2.22	L-R	123	0.00
109	3.23	0.00	L-R	124	0.64
			2 MA		
130	4.98	0.00	L-R	125	0.87
131	1.69	2.09	L-R	126	0.00
132	0.95	0.00	L-R	127	0.00
133	0.00	8.44	L-R	128	0.00
134	0.00	9.48	L-R	143	0.89
135	2.88	6.35	R-L	142	0.84
136	9.47	5.80	R-L	141	0.00
137	2.27	6.45	R-L	140	0.00
138	0.00	4.69	R-L	144	0.59
139	1.38	2.80	R-L	145	0.00

$\overline{X}$ ESCAPE LATENCIES FOR TRIALS 6-75

Barrier 2

1 MA

Rat No.	SDL	SDR	Order	Rat No.	Control
44	3.02	2.35	L-R	22	0.00
66	10.48	3.29	L-R	27	1.96
67	5.02	3.68	L-R	28	0.00
68	5.53	4.41	L-R	29	0.93
69	6.11	8.49	R-L	30	2.47
70	4.55	3.10	R-L	31	0.78
75	7.93	2.64	R-L	32	0.75
74	0.00	1.42	R-L	25	0.00
76	3.02	1.28	L-R	46	1.46
78	4.21	3.92	L-R	62	3.22

2 MA

70	6.32	0.00	R-L	63	4.97
80	2.11	2.74	R-L	71	0.73
81	3.72	3.86	L-R	73	0.85
86	5.02	7.87	L-R	72	0.00
85	3.34	0.00	R-L	82	0.00
88	11.28	48.04	R-L	83	0.00
89	3.41	2.68	L-R	87	0.00
90	3.71	3.46	L-R	84	1.24
91	12.30	3.56	R-L	93	0.00
92	0.00	2.61	R-L	94	0.00

$\overline{X}$ ESCAPE LATENCIES FOR TRIALS 6-75

Barrier 3

1 MA

Rat No.	SDL	SDR	Order	Rat No.	Control
146	2.12	1.83	L-R	98	1.56
147	6.79	0.00	L-R	99	1.53
148	2.28	0.00	L-R		0.92
149	2.18	4.48	L-R	160	0.73
110	1.72	1.66	L-R	161	0.81
111	2.69	4.89	R-L	116	1.42
152	5.07	1.51	R-L	117	1.21
153	2.44	2.51	R-L	118	6.00
158	3.85	18.45	R-L	119	1.29
159	1.25	1.62	R-L	120	1.29

2 MA

162	2.09	2.76	R-L	225	0.00
163	3.30	1.97	R-L	224	0.85
164	1.34	1.85	R-L	222	0.00
165	0.00	27.37	R-L	221	0.00
207	2.21	2.58	R-L	213	0.88
208	4.58	2.07	L-R	113	0.00
209	1.41	0.41	L-R	112	0.00
210	0.00	1.57	L-R	155	0.00
211	1.23	3.52	L-R	156	0.00
212	1.51	1.23	L-R	154	0.00

$\overline{X}$ ESCAPE LATENCIES FOR TRIALS 6-75

Barrier 4

1 MA

Rat No.	SDL	SDR	Order	Rat No.	Control
214	2.45	1.45	L-R	219	0.86
215	21.28	2.47	L-R	223	0.00
216	0.00	2.10	L-R	227	0.64
226	0.00	0.00	L-R	235	0.79
218	2.17	3.08	L-R	228	0.89
254	0.00	3.34	R-L	244	1.27
230	5.10	3.48	R-L	245	0.00
231	8.12	1.11	R-L	255	0.00
233	3.56	2.15	R-L	258	0.00
232	2.32	2.23	R-L	259	0.00
			2 MA		
236	2.34	2.26	R-L	229	1.79
256	3.31	1.99	R-L	251	0.00
238	2.54	3.92	R-L	242	0.76
239	1.12	1.26	R-L	243	1.42
250	3.48	4.45	R-L	247	0.00
246	2.98	0.00	R-L	240	5.30
248	1.74	0.00	L-R	241	0.82
249	0.00	0.00	L-R	253	0.00
252	0.88	9.59	L-R	260	4.16
257	1.79	2.01	L-R	261	0.00

$\overline{X}$ ESCAPE LATENCIES FOR TRIALS 6-75

Barrier 5

1 MA

Rat No.	SDL	SDR	Order	Rat No.	Control
170	38.96	5.00	L-R	166	1.38
171	2.43	0.00	L-R	167	0.00
172	2.68	2.50	L-R	168	0.00
173	2.26	2.67	L-R	169	0.00
174	2.19	0.41	L-R	179	0.00
175	2.19	3.93	R-L	186	0.00
176	1.31	1.35	R-L	187	0.00
177	2.23	2.22	R-L	197	0.00
181	0.96	4.02	R-L	205	0.00
178	1.50	2.32	R-L	206	0.00
			2 MA		
182	0.00	0.00	R-L	184	0.00
188	5.26	0.75	R-L	185	0.61
189	0.00	3.42	R-L	183	1.51
190	0.00	0.81	R-L	198	5.22
191	1.30	0.00	R-L	199	0.00
192	0.00	8.13	L-R	200	0.00
193	1.70	0.00	L-R	201	1.07
194	1.25	0.00	L-R	202	0.80
195	2.16	0.00	L-R	203	0.00
196	0.00	5.70	L-R	204	0.00